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EXPERIENCE WITH COLLAPSE THERAPY FOR PULMONARY TUBERCULOSIS IN THE FIFTH AND SIXTH DECADES*

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COLLAPSE therapy has become indispensable in the treatment of chronic pulmonary tuberculosis. Indications and contraindications have been well established upon which to base selection of cases, in keeping with varied clinical and pathologic findings. Technique has been steadily improved so that morbidity and mortality have been reduced. It cannot be said, however, that the millennium has been reached, that the selection of risks is infallible, that technique has been entirely stabilized and perfected, nor that results now attained are all to be desired. There has been broadening of the base of operations so that, whereas originally unilateral cases only were considered suitable for collapse therapy, now bilateral disease is freely treated. At first only individuals between the ages of 20 and 40 years were thought to be good prospects and good risks. Now there is hardly any upper age limit. There has been, however, comparatively little discussion and few reports of results obtained in patients of the fifth and sixth decades, that is, above the age of 40 years. At this age, when two-thirds of life for the average man has been passed, and life has nearly ended for the tuberculous patient, have the various collapse operations, artificial pneumothorax, phrenic nerve paralysis, pneumolysis, and thoracoplasty been successful? Do the results justify wider or more-restricted employment? Diminishing returns are naturally to be expected. To what degree do these patients respond? Can special criteria be established for proper selection of cases in this age group?

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To seek an answer to some of these questions, we have reviewed a series of 154 patients between the ages of 40 and 60 years, for whom collapse therapy has been undertaken in various ways. This treatment has been given for the most part at the Tuberculosis League Hospital, Pittsburgh, in the last ten years, and the Veterans' Facility at Aspinwall, Pa., in the last two years. Detailed statistical analysis of these case records will be found in the tables.

TABLE I

154 PATIENTS AGED 40 TO 60 YEARS

	ARTIFICIAL PNEUMOTHORAX	PHRENIC NERVE OPERATION	THORACOPLASTY	PLOMBAGE	SUMMARY
No.	78	73	17	3	171
Age	40-54	40-60	41-54	41-57	40-60
	4>50	12>50	1>50	2>50	19>50
Sex	M. 62	M. 62	M. 12	M. 3	M. 139
	F. 16	F. 11	F. 5		F. 32
Duration of disease	6 mo.-14 yr.	6 mo.-18 yr.	1-20 yr.	6 mo.-3 yr.	6 mo.-20 yr.

MULTIPLE OPERATIONS

Artificial pneumothorax followed by phrenic nerve operation	6
Artificial pneumothorax followed by thoracoplasty	5
Artificial pneumothorax followed by phrenic nerve operation and thoracoplasty	1
Artificial pneumothorax followed by plombage	1
Thoracoplasty followed by phrenic nerve operation	3
Phrenic nerve operation followed by plombage	1
	17

OPERATIONS PRIOR TO 40 YEARS OF AGE

Artificial pneumothorax followed by phrenic nerve operation	1
Artificial pneumothorax followed by thoracoplasty	1
Phrenic nerve operation followed by artificial pneumothorax and thoracoplasty	1
	3

It is appreciated that this is a comparatively small group upon which to base conclusions, but at least it is large enough to trace trends in the reaction of these older patients to collapse treatment. For the purpose of recording the results condensed, National Tuberculosis Association nomenclature has been utilized—cases are classified as arrested, improved, stationary, worse, and dead. The basic criteria for establishing the proper status have been negative sputum and closure of cavities, rather than symptomatology, such as general well-being, gain in weight, loss of temperature, diminution of cough and sputum. When they have been considered arrested, they have been free from any signs of activity for at least a year and a half. Improved cases must have had marked diminution or closure of cavity and have fairly consistently negative sputum. Included in this category are those patients who may have negative sputum and closed cavities, but have not been symptom-free

for at least eighteen months. Many in the stationary group manifested general improvement in symptoms but did not have the two essential desiderata for advanced standing.

In the group of 154 patients, 19 were more than 50 years old. There were 122 males and 32 females. Upon these patients 171 operations were performed: 16 had two types of collapse therapy, and one individual had three different forms; 3 patients had had one operation before the age of 40 years, but these operations are not included as part of the total 171.

The tuberculosis had been recognized and treated for periods of six months to twenty years. In many of the patients with the shorter histories clinical and x-ray findings pointed to a much longer duration of infection. In at least half the cases the disease had been admittedly of more than five years' standing.

Pathology.—Statistics are found in Table II. About 50 per cent of the patients had productive fibrous lesions, 25 per cent productive and exudative combined, and 25 per cent predominantly exudative. There were

TABLE II

PATHOLOGY

			ARTIFICIAL PNEUMOTHORAX	PHRENIC NERVE OPERATION	THORACOPLASTY	PLOMBAGE
Productive	73	48.0%	41	29	8	2
Exudative	36	23.0%	25	10	2	
Productive and exudative	45	29.0%	12	34	7	1
Moderately advanced	41	26.0%	21	19	4	
Far advanced	113	73.0%	57	54	13	3
Unilateral	24	15.5%	15	11	2	
Bilateral	26	16.5%	11	14	1	
Unilateral with cavity	40	26.0%	22	13	8	
Bilateral with cavity	12	8.0%	3	8	1	1
Bilateral with unilateral cavity	52	34.0%	27	27	5	2
Diseased area equivalent to ⅓ lung	11	7.0%	2	9		
Diseased area equivalent to ½ lung	15	9.5%	7	7	2	
Diseased area equivalent to ⅔ lung	25	15.0%	15	8	2	
Diseased area equivalent to one lung	37	23.0%	27	12	8	
Diseased area equivalent to more than one lung	66	49.0%	27	37	5	3
Cavity, apical or upper lobe	94	61.0%	47	42	15	3
Cavity, base or lower lobe	7	4.5%		7		
Cavity, multiple	5	3.3%	2	2	2	
Total	106	69.0%	49	51	17	3

no minimal cases. About 25 per cent were moderately advanced, and the remainder far advanced. Nearly 50 per cent of the patients had bilateral lesions. In about 50 per cent, more than the equivalent of one lung was diseased. Cavitation was present in 106, or 69 per cent of the group. In about 90 per cent the cavity was apical or in the upper lobe.

Five patients had more than one demonstrable cavity and 4 bilateral cavitation. For the purpose of this paper a cavity was counted only when its diameter was at least 1.5 cm. Twice, cavities were 10 cm. in diameter, in 6 cases more than 5 cm. About 50 per cent of the cavities were thick walled on x-ray appearance; about 90 per cent were placed in the midst of lesions of the fibrosing type.

Clinical results in bilateral disease were in inverse proportion to unilateral. For instance in the arrested class 17 per cent were bilateral as opposed to 83 per cent unilateral; in the mortality group 77 per cent were bilateral and 23 per cent were unilateral. The results on the basis of type of lesion showed that 75 per cent of the arrested group had productive tuberculosis as opposed to the exudative, but unexpectedly 75 per cent of mortality was in the productive class.

Only one patient in the entire group had extrapulmonic tuberculosis, this a mediastinal lymph gland involvement. One patient only developed empyema, a pyogenic affair following spontaneous perforation.

From an economic standpoint the earning capacity of this entire group was zero. They had been family or institutional wards for variable periods of time, and all but 7 had positive sputum and were therefore infection carriers. Most of the group had been given medical treatment at home or in the hospital with indifferent results, and were without exception doomed to invalidism and to die of the disease. For them, collapse therapy had everything to gain and nothing to lose.

In prescribing collapse treatment a primary procedure was advised which seemed to meet the needs of the individual case whether pneumothorax, phrenic nerve operation, thoracoplasty, or plombage. In general, pneumothorax was preferred, but in at least one-third of the cases it was impossible to induce the pneumothorax, so that a less desirable substitute was utilized. A number who refused our choice of primary procedure accepted a less efficient one.

Clinical results are recorded in Tables III and IV. They represent treatment carried out for 114 patients in a period of six months to two years, for 25 in a period of two to five years, for 15 in a period of five to ten years. It is obvious that, for the most part, too short a time has elapsed to establish a final rating, but the trend and ultimate outcome have been indicated. Undoubtedly some patients have been recorded as improved who will proceed to recovery; a few now stationary will progress to the improved class, or even to recovery, particularly if supplementary treatment is undertaken. At the same time individuals now perfectly well may have a relapse or recurrence of the disease and those stationary may die. In round figures for this series it is to be expected that not more than 20 per cent will ultimately be well and able to do some work; that 25 per cent will be improved to the degree that they will have negative sputum and be able to do part time work; that 25 per

cent will be invalids for a variable number of years until they die of the disease; that the remaining 30 per cent have already died or soon will die. Compared with our results of collapse therapy for the age group of 20 to 40 years, which are, roughly, arrested, 34 per cent; improved,

TABLE III

CLINICAL RESULTS

NO. OF PATIENTS			A.P.	PHR.	THOR.	PLOM.	A.P. AND PHR.	A.P. AND THOR.	A.P. AND PLOM.	A.P., PHR. AND THOR.	PHR. AND THOR.	PHR. AND PLOM.
Arrested	23	15.0%	8	12	1		1			1		
Improved	45	29.2%	18	22	3	1				1	1	
Stationary	35	22.9%	17	12			2	2	1		2	1
Worse	7	4.5%	4	3								
Died, tuberculous	38	24.5%	18	12	3		2	2		1		
Died, non-tuberculous	6	3.9%	2	1			1	1		1		
Total			67	62	7	1	6	5	1	4	3	1

Time elapsed since treatment	114	6 mo.—2 yr.
	25	2—5 yr.
	15	5—10 yr.
Arrested cases, patients have remained well		6 mo.—2 yr. 12
		3 yr. 2
		4 yr. 2
		5 yr. 4
		6 yr. 1
		8 yr. 2

A.P. = Artificial pneumothorax.
Phr. = Phrenic nerve operation.
Thor. = Thoracoplasty.
Plom. = Plombage.

TABLE IV

CLINICAL RESULTS BASED UPON OPERATION

	ARTIFICIAL PNEUMOTHORAX		PHRENIC NERVE OPERATION		THORACOPLASTY		PLOMBAGE	
Arrested	10	12.8%	14	19.1%	2	11.8%		
Improved	20	25.6%	25	35.0%	5	29.5%	2	66.6%
Stationary	22	28.2%	16	22.4%	4	23.6%	1	33.3%
Worse	4	7.8%	3	4.2%				
Died, tuberculous	22	25.6%	15	19.3%	6	35.1%		
Died, nontuberculous	5	6.4%	3	4.2%	2	11.7%		
	83		76		19		3	

22 per cent; stationary, 14 per cent; died, 30 per cent; there is a difference of 10 per cent to 15 per cent in less favorable response. Stated in another way, an individual before 40 years of age has one chance in three of recovery, after 40 one chance in five after he has had collapse treatment. At either age he has one chance in four of being improved materially.

Artificial Pneumothorax.—Statistics are found in Table V. Induced in 78 individuals, artificial pneumothorax was carried to completion in only 20 patients (25 per cent). It was discontinued because of unsatisfactory collapse in 12 (15 per cent) and for other reasons in 28 (35 per cent). Ten per cent of the patients refused to continue the treatment after it was started. Based on the number of operations arrest was obtained in 16.6 per cent, improvement in 24.5 per cent, no benefit in 32 per cent, and mortality in 26.9 per cent. These figures are considerably less favorable than one might expect to find in the pneumothorax group.

The amount of collapse varied considerably. In one-fifth of the cases it was less than 10 per cent. The high percentage of inefficient collapse is to be accounted for by the prevalence of a preexisting pleuritis.

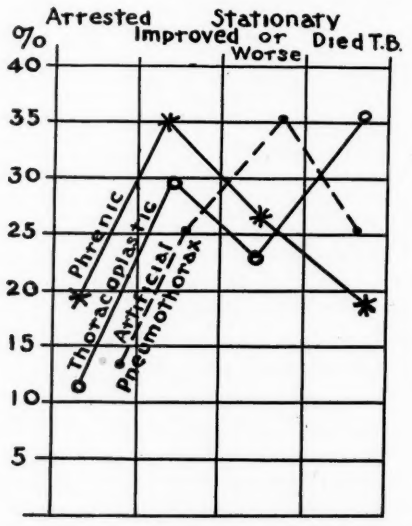

Fig. 1.—Graph of clinical result. For percentage, see Table IV.

Treatment was maintained for periods of three months to six years. The figures show quite well that success in closing cavities, and making sputum negative by pneumothorax in this age group, is in direct proportion both to the degree of the lung collapse and to the length of time the pneumothorax is continued.

It was noted that effusions did not develop any more readily than in younger age groups. There were only ten instances. Spontaneous perforation occurred in two patients. In one, empyema followed and required open drainage followed by thoracoplasty. In the second case perforation occurred soon after the first induction of air with fatality in twenty-four hours. There were no instances of so-called pleural shock. Twelve patients were given artificial pneumothorax to afford sympto-

matic relief; for example, to control bleeding, without much expectation of success, and in fact with little realization.

TABLE V

TREATMENT BY ARTIFICIAL PNEUMOTHORAX. SEVENTY-EIGHT CASES, ALL UNILATERAL

Treatment completed	20
Treatment discontinued, unsatisfactory collapse	12
Treatment in progress	20
Treatment discontinued before completion	26
Patient refused further treatment	8
Fluid	7
Spontaneous pneumothorax	1
Pleuritis	5
Extension to opposite lung	5

DEGREE OF COLLAPSE	NO.	A	I	S	W	D	SPUTUM (26)	CAVITY (46)		
							+ → 0	CLOSED	SMALLER	NO EFFECT
Poor	12			8		4	2			4
10%	13		1	5	2	5			1	7
20%	10		3	4		3	6		2	7
30%	14	2	5	2	2	3	3	1	2	3
50%	12	2	5	1		4	6	2	4	4
75%	17	6	6	2		3	9	6	3	
Total	78	10	20	22	4	22	26 (33.3%)	9 (19.5%)	12 (26.0%)	25 (54.5%)
DURATION ARTIFICIAL PNEUMO-THORAX	NO.	A	I	S	W	D	SPUTUM	CAVITY		
							+ → 0	CLOSED	SMALLER	NO EFFECT
to 3 mo.	15		1	8		6				4
3-6 mo.	23		8	6	4	5	3			5
6-12 mo.	19	1	7	7		4	10	1	5	10
12-18 mo.	8	2	3	1		2	4	4	3	4
2 yr.	8	4				4	6	2	2	2
3 yr.	3	1	1			1	2	1	2	
4 yr.	1	1					1			
6 yr.	1	1						1		
Total	78	10	20	22	4	22	26 (33.3%)	9 (19.5%)	12 (26.0%)	25 (54.5%)

A = arrested.
I = improved.
S = stationary.
W = worse.
D = died.

Phrenic Nerve Operations.—Statistics of this series of 73 operations are found in Table VI. The relatively high number of exereses was due in some degree to uncertainty as to the future control of the patient. Whereas the phrenic nerve operations were done as a primary procedure in all but 6 of the group, they were second choice and a poor second in 15, owing to patients' refusal of a more desirable pneumothorax or thoracoplasty. Based on the number of operations 18 per cent of the cases were arrested and 33 per cent improved. In all, fixation of the diaphragm was obtained. The degree of the rise varied from none to 4 inches, and seemed comparable to that obtained in younger age groups. Success in closing cavities and rendering sputum negative was in direct

proportion to the height of the elevation of the diaphragm. Cavities were closed in 15 per cent and made appreciably smaller in 20 per cent. This is a distinctly smaller return than in the younger age groups. Twice, a diaphragmatic paralysis was helpful in supplementing a pneumothorax about to be lost, and twice as adjuvant to upper thoracoplasty. In 15 cases the operation was undertaken for hemoptysis, which was completely controlled in 5 instances, and partially in 5 others.

TABLE VI

PHRENIC NERVE OPERATIONS

Number operations	73	
Crush	25	(no recrushes)
Exeresis	48	
Primary procedure	67	
Secondary to artificial pneumothorax	3	
Secondary to thoracoplasty	3	
Followed by thoracoplasty	1	
Followed by plombage	1	
As second choice	6	
Expectation of favorable result	25	34.2%
Expectation realized	18	26.6% (or 72% realization)

RISE OF DIAPHRAGM	NO.	A	I	S	W	D	SPUTUM + ↓ −	SPUTUM − ↓ −	SPUTUM + ↓ +	CAVITY (50) CLOSED	SMALLER	LARGER	NO EFFECT
None	11		1	5	1	4	2	1	8	1	1		6
1″	26		9	6	1	10	5	1	20		6	3	12
2″	22	6	11	3	1	1	9	1	12	2	3		9
3″	11	7	3	1			6	2	3	5			1
4″	3	1	1	1			1		2		1		
Total	73	14	25	16	3	15	23	5	45	8	11	3	28

Sputum	23 (33.8%)	became negative
	45 (66.2%)	remained positive
Cavity	8 (14.3%)	closed
	11 (20.3%)	smaller
	3 (5.5%)	larger
	28 (60.0%)	not affected
Hemoptysis	15 cases	
	10 stopped or less	

A = arrested.
I = improved.
S = stationary.
W = worse.
D = died.

Thoracoplasty.—Statistics are found in Table VII. Thirty-four operations were performed upon 17 patients: as a primary procedure in 9 patients; after pneumothorax had failed in 5, and after pneumothorax and phrenic exeresis together were ineffective in 3. In 4 patients, the operation as planned was not finished because of activation or spread of preexisting pulmonary lesions. In none was new territory immediately invaded. Arrest was obtained in 2, or 11 per cent of the total group, improvement in 6, or 35 per cent. Three in the improved class have closed cavities and negative sputum and are reasonably certain to re-

cover, but they have been operated upon within a year and cannot be classified at this time as certainly arrested. Should they remain well, the arrested group would total 30 per cent. The mortality has been high, about 30 per cent, if 2 late deaths from nontuberculous disease are excluded. The operation mortality, based upon the total number of

TABLE VII

THORACOPLASTY

Total number of patients	17	Posterior	33	
		Lateral	1	
Total number of operations	34			
		One stage	5	
Thoracoplasty, primary	9	Two stage	7	
		Three stage	5	
Thoracoplasty, secondary to artificial pneumothorax	5	Right	10	(5 dead)
		Left	7	(1 dead)
Thoracoplasty, secondary to artificial pneumothorax and phrenic nerve operation	3			
Thoracoplasty completed	13			
Thoracoplasty not completed	4			
Collapse good	10			
fair	3			
incomplete	4			
Sputum positive in	17	became negative	6	
Cavities in	17	closed	6	
		smaller	2	
Hemoptysis in	3	became less	3	

MORTALITY

Total	8/17	47.2%
Immediate, attributable to operation	2	11.8%
Delayed, attributable to operation	3	17.7%
Delayed, from tuberculosis	1	5.9%
Delayed, not from tuberculosis	2	11.8%
Operation mortality (within 48 hr.)	2/34	5.9%

Cause of death:

1. Hemorrhage, subclavian artery, 16 hr. after 1st stage.
2. Pulmonary edema, 36 hr. after 2nd stage, probably cardiac.
3. Cardiac failure, 1 wk. after 3rd stage.
4. Progressive tuberculosis, 1 mo. after 2nd stage.
5. Progressive tuberculosis, 2 mo. after 2nd stage.
6. Mesenteric thrombosis, 10 mo. after 3rd stage, tuberculosis arrested.
7. Depression psychosis, 6 mo. after 3rd stage, tuberculosis improved.
8. Tuberculosis contralateral lung, 4 yr. after 3rd stage.

Preoperative risk	good 8	Died	2	25%
	fair 7		5	71%
	poor 2		1	50%

operations, has been about 6 per cent. Both of these figures are distinctly higher than in the younger patients. After careful preoperative study 8 of the patients were considered to be good risks, 7 fair, and 2 poor. Mortality percentages in these three classes were 25, 30 and 50 per cent. There was a preponderance of fatality in right-sided thoracoplasty.

Plombage.—Statistics are found in Table VIII. This operation of extrapleural pneumolysis with paraffin pack was undertaken in 3 patients when apicolysis was refused. The first was a primary procedure to close a cavity in a 57-year-old man with bilateral apical disease. X-ray appearance at the end of five months indicated closure of the cavity and improvement in the contralateral lung. Report from this man at the end of ten months is of general improvement, infrequent cough, scant negative sputum, and gain in weight. The second patient is a man 52 years old with bilateral disease who has carried a pneumothorax in the right side for eighteen months without marked improvement and still with positive sputum. Plombage was used in the left apex to close there a comparatively small persistent cavity. Since the plombage, the sputum

TABLE VIII

PLOMBAGE—3 CASES

AGE	PATHOLOGY	TREATMENT	SPUTUM	CAVITY	CLINICAL RESULT
57 yr.	Productive Far advanced Upper ⅓ bilateral with cavity, right Empyema Thickened pleura	Plombage Right apex	+ → 0	Right closed Left smaller in 2 mo.	Improved
52 yr.	Productive Exudative Far advanced Total left lung Upper lobe, right Bilateral apical cavitation	Artificial pneumothorax, left 75% collapse Plombage, right	+ → + + → +	Smaller in 6 mo. Cavity apparently closed	Stationary
41 yr.	Productive Far advanced Bilateral Upper ½ right Upper ½ left with cavity	Phrenic nerve crushed, no effect, left Plombage, left	+ → + + → 0	Larger in 6 mo. Closed in 8 mo.	Improved

has become negative at times. He is losing his pneumothorax so that the future result is uncertain. One adhesion which just now may be a factor hastening the reexpansion will be divided intrapleurally. The third patient is a 42-year-old man with bilateral upper lobe tuberculosis and a sizable cavity on the left side. He refused thoracoplasty and artificial pneumothorax. The phrenic nerve was crushed as a substitute, without benefit, the cavity really becoming larger. Eight months ago he consented to a plombage. The cavity is now closed and the sputum is negative. Two patients, therefore, have been markedly improved, while the third is classed as stationary.

DISCUSSION

On the basis of this small series of cases we feel that the results obtained more than justified the use of collapse therapy after the age of 40 years. Response will be at a lower level, recoveries will be fewer by

10 to 15 per cent than in the earlier decades, and the mortality higher, but possibly not appreciably more than would be expected because of the natural progress of the disease, and the higher general mortality rate of advanced years. As a group we have found these older patients less faithful in pursuing the postoperative treatment that is essential to complete success. In the pneumothorax series, particularly, maximal results were not attained because patients were unwilling to carry on to completion.

Any danger with the use of pneumothorax and diaphragmatic paralysis is concerned with collapsing good lung parenchyma and sacrificing vital capacity essential to these older patients. In the case of pneumothorax the situation is controllable, but after phrenic interruption it is not remediable until the nerve regenerates and the muscle functions again, in a space of from three months to two years.

The operative hazard of artificial pneumothorax, of phrenic nerve operations, of extrapleural and intrapleural pneumolysis is not greater after 40 years of age than before. Postoperative complications are not more frequent nor different. In thoracoplasty only is there a greater risk than in the earlier age groups. This is attributable, it seems to me, to the following factors:

1. *Lowered threshold of resistance and vitality.* This is a rather intangible consideration but I believe it is a real one. Older individuals to a larger degree are more susceptible to pyogenic infection, to slower healing of the tissues, and to easier activation and spread of the preexisting tuberculous lesions.

2. *Pathology in the lungs.* This differs little in its general characteristics from the younger groups, but there is certainly a greater amount of pleuritis interfering not only with the induction of pneumothorax but with collapse of the lung. Cavities are apt to be thicker walled. The important factor is the tendency to fibrosis and emphysema in the good or nontuberculous lung, which deprives the individual of vital capacity and respiratory function in excess of what is to be expected on the basis of x-ray examination and vital capacity tests, and builds up an undesirable increased intrapulmonary pressure.

3. *Condition of the cardiorenal-vascular system.* There is a definite tendency for these individuals past 40 years of age to develop postoperative nephritis. The older the individual the greater is the probability of his having developed, if not actual palpable sclerosis in his arteries and hypertension, at least, loss of resilience. The heart is the enigma. We are certain that the heart of the tuberculous patient over 40 years of age is not nearly as efficient as the heart of a patient under 40 years of age and is not as able to stand the strain of a thoracoplasty. Tests of functional capacity, such as the two step test, do not record it. Physical examinations show no evidence of myocarditis and valvular lesions. Electrocardiograms are negative.

The circulatory disturbance which sometimes follows thoracoplasty is a complex problem, and the course of events is not entirely clear. Presumably in most cases the cardiac fatigue and failure come primarily from oxygen deficit of pulmonary origin, due to increased intrapulmonary pressure from mechanical bronchial obstruction or some neurocirculatory disturbance. The net result is a pulmonary engorgement followed by pulmonary edema to which the heart itself contributes as muscle function fails, so that a vicious circle is established. The end result is cardiac failure and death. Occasionally there is also failure of the medullary centers from the intensity of the anoxemia. One cannot but feel that at times the mainspring of the circulatory disaster is extrapulmonic in origin, that it results from increased mediastinal pressure set up after rib removal. The pulmonary veins are compressed so that blood flow from the lung into the left auricle is impeded, and at the same time the muscle receives a lessened and, it may be, deficient blood supply. This retardation of the pulmonary circulation eventually causes anoxemia.

The clinical picture of this catastrophe is striking and distinctive. For a period of several days to a week following a thoracoplasty everything is serene. Then a progressive dyspnea develops, at first without cyanosis. There is no fever. Cardiac action remains regular, and at the start is only slightly increased in rate. Moisture is found at the bases of the lungs, particularly of the good lung. It spreads rapidly and becomes ultimately a general pulmonary edema. Then the pulse rate rises to 130 and may reach 160. Blood pressure which has been at a satisfactory level drops quickly to 50 or 60 systolic. Death comes in from thirty-six hours to five days after the onset. Oxygen, transfusions, and medication are of no avail. These patients are doomed. At autopsy the heart does not look grossly abnormal though the muscle may appear soft and flabby. Microscopically, cloudy swelling of the muscle fibers may be found, but not myocarditis. There is no dilatation of the heart, no pneumonitis, no spread of tuberculosis. The operation wound is found to be in perfect condition. There is no definite pressure to be observed on the base of the heart by the collapsed lung or the chest wall nor mediastinal displacement. The pity is that it cannot be foreseen which patient will develop this circulatory collapse. At times those who are apparently the best risks die in this way. We are sure that the older patients are more prone to this development.

In the light of our experience we would consider that important contraindications to thoracoplasty are low vitality, cardiac symptomatology, subjective or objective, nephritis, bronchitis, and emphysema in the contralateral lung and low total vital capacity equivalent to less than 1,000 c.c.

In spite of increasing the risk of mortality by multiple operations, the number and length of rib resections should be less than in earlier age groups. We do not think that an absolute rule can be established. Ordinarily it is safe to remove parts of two to two and one-half ribs at one stage including the transverse processes. We believe it advisable not to go farther than the anterior axillary line in the primary posterior operation. If needful to establish a satisfactory collapse, these anterior segments can be removed more safely at a subsequent anterolateral operation. On the other hand, we believe it is futile to do too little at one time and not remove sufficient rib lengths to obtain a collapse. These patients may survive the thoracoplasty but they are not cured and are little better off than if they had not been operated upon at all.

More than the usual amount of care must be exercised in the anesthesia. For the most part it would be ideal to operate with only local anesthesia. We have found this to be a considerable strain upon the morale of the patient, who may tolerate it for the first stage but will refuse it subsequently. We have never fancied general inhalation anesthesia in pulmonary tuberculosis, but prefer to give our patients a dose of from 70 to 80 mg. of avertin to supplement the local anesthesia. Usually individuals who are so anesthetized are awake and coughing within one to two hours if they are given injections of ephedrine and caffeine as they leave the operating room. It has been our habit in the last year or two to place the patients in bed with the foot elevated so as to aid clearing of the lower lobes by gravity until they are able to cough vigorously.

CONCLUSIONS

1. *Collapse therapy* for patients in the fifth and sixth decades is of great value. A sizable number will be restored to health and earning capacity who are otherwise doomed to chronic invalidism and to be an economic burden, as well as potential spreaders of infection. Expectations are not realized to the same degree, and returns are less satisfactory than in earlier age groups. Many successes, however, come when least looked for. It is this which justifies a collapse operation somewhat as an experimental procedure when all the signs point to futility.

2. *Artificial pneumothorax* will be possible to the point of satisfactory collapse in a much smaller percentage after 40 years of age, and selective collapse is less likely to be obtained because of the greater frequency of adherent pleuritis. Pneumothorax should be tried as a primary procedure before resorting to the other methods on account of its relative safety, but either it should be discontinued without delay if a satisfactory collapse has not taken place, or it should be supplemented by other types of operation.

3. *Phrenic nerve operations* show the best returns of all the methods. In cases with basilar pleuritis on account of fixed diaphragm little is to

be expected by phrenic paralysis, nor is it to be expected that very thick-walled cavities in the midst of extensive productive lesions, or close to the periphery of the lung, will be frequently influenced.

4. *Plombage* is really a valuable adjuvant in this age group, particularly in bilateral disease, and may easily accomplish at minimal risk what a thoracoplasty would do. We do not know how innocuous this paraffin pack will prove to be. In our observation the pleura has proved to be thick in apical disease, and seems to be well fortified against perforation and its attendant complications. On the basis of experience with individuals below the age of 40 years, we believe plombage is a valuable operation for cavitation also in the middle and lower lung fields.

5. *Thoracoplasty* is needed, and should be utilized but with greater precaution and hesitancy than in the earlier age groups. The fact that there will be mortality due to the operation, and that the rate will be higher than in youth should not be a deterrent but it should make one more willing to try other procedures first. There can be no question as to its value in being the most positive and expeditious form of collapse.

Acknowledgment is due Dr. George E. Martin, medical director of Leech Farm, Dr. G. C. Emery of the Veterans' Facility, Aspinwall, Pa., and Dr. L. H. Hetherington, Resident Physician of the Tuberculosis League Hospital, for their aid in the preparation of this paper.

PARTIAL THORACOPLASTY FOR PULMONARY TUBERCULOSIS

With a Suggested Plan of Operation Including Preliminary Anterior Chondrocostectomy, Together With a Review of Results*

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PHYSICAL and physiologic rest have become bywords in the treatment of pulmonary tuberculosis. How these ends may be attained by the simplest and most effectual means has been the absorbing interest of many minds engaged in the care of the tuberculous. No agent has excelled an adequate pneumothorax in achieving a satisfactory pulmonary collapse in which respiratory action is reduced to a minimum and the walls of cavities in the lung are approximated. Moreover, it achieves this purpose by exacting no big toll from the patient. It does not ask that the patient be a first class risk; its *only* requisite is the presence of a free pleural space. Many cases fail to meet this single requirement and must in consequence accept the substitute measure of thoracoplasty which demands a great capacity on the part of the patient for the tolerance of violence.

That many patients proved unequal to the extortions of the formidable procedure of complete posterior thoracoplasty, surgeons learned to rue in their mortalities; an additional cause for grave concern was the large number of instances in which the accomplishments of thoracoplasty failed to justify its performance.

No substitute has been found for a free pleural space which would widen the field of usefulness of pneumothorax except in those instances where a few string or cordlike adhesions restrain collapse and can be dealt with by intrapleural pneumonolysis. Surgeons have learned, however, to effect more complete collapse of the lung with thoracoplasty, at the same time exacting less from the patient. This contribution of thoracic surgeons during the past decade has been a big boon to patients with pulmonary tuberculosis needing collapse therapy. Now, because of the better results achieved with less risk, patients more readily submit to thoracoplasty.

MODERN OPERATION OF THORACOPLASTY

When excision of the upper ribs was instituted as the first procedure of thoracoplasty, and recognition became general of the necessity for mul-

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tiple stage operations, the modern operation had its inception. More and more the tendency appears to be toward the partial or selective thoracoplasty with complete excision of the upper three ribs and sacrifice of shorter segments of succeeding ribs in subsequent operative procedures. Primary excision of the transverse processes has improved the collapse achieved in the posterior portion of the lung, in which cavity formation is most frequent—thus securing a greater number of primary closures of pulmonary cavities. The sacrifice of segments from more than six or eight ribs is limited to those patients for whom the partial operation is inadequate. Satisfactory obliteration of pulmonary cavities with minimum sputum which is negative on examination for tubercle bacilli, and sufficient collapse of the thoracic cage to maintain pulmonary compression, are the considerations which determine whether rib removal has been adequate. Amputation of the base of the scapula and the employment of anterolateral thoracoplasty as a complementary operation are measures which have contributed to the attainment of an adequate pulmonary collapse without removal of segments of as many ribs as was customary in the Wilms-Sauerbruch type of thoracoplasty.

OUR PLAN OF OPERATION

The plan of operation described here has been followed as the procedure of choice since March, 1934, in the performance of thoracoplasty at the University Hospital. It was the thought of the senior writer of this paper that preliminary anterior chondrocostectomy of the upper ribs would not only improve the collapse but facilitate the performance of the posterior operation as well. Our experience of somewhat more than three years with this plan of operation has confirmed these impressions.

At first, one or two short parasternal transverse incisions were made in the second and fourth interspaces through which the entire costal cartilage and short segments of rib were removed from the second through the fourth or fifth rib. Soon, however, it was found advantageous to include excision of the cartilage of the first rib, facilitating thereby considerably complete excision of the first rib from behind. It was also found that the time interval between the anterior chondrocostectomy and the second posterior operation was usually so long that complete rib removal beyond the third rib was not made easier by the preliminary operation. Furthermore, the closure of posteriorly situated pulmonary cavities was not enhanced by complete rib removal beyond the third rib. In consequence, the skin incision employed was modified to facilitate the removal of the first rib cartilage. A preliminary note concerning this operative procedure was published in 1935 (Carlson).

ANTERIOR CHONDROCOSTECTOMY

This procedure has been done in most instances under infiltration of the skin and subjacent tissues with 1 per cent procaine solution. Occasionally cyclopropane inhalation anesthesia has been employed. The latter agent has been used solely in the posterior operations. The second and third costal cartilages can be readily removed through a short transverse incision, and with proper retraction the first rib cartilage can also be nicely exposed through this incision. An oblique parasternal incision affords the most direct approach but of course leaves a more prominent scar. These incisions are shown in Fig. 1. In this procedure the first, second, and third cartilages are excised with simultaneous removal of the most distal portion of the second and third ribs. A short segment of the fourth

cartilage may also be removed as indicated in Fig. 1. Not alone is the entire cartilage removed, but in addition a short segment of the rib is removed, for, by so doing, the final separation of the most anterior portion of the rib from its periosteal bed is found to be much easier at the time of the first posterior operation. Excision of only 1 or 2 cm. of the anterior portion of the rib at the time of the chondrocostectomy is sufficient. Our present usual practice is to make a single short transverse incision over the second rib, through which the costal cartilages of the first, second, and third ribs may be removed. At the same time, the rib is detached well posteriorly from its bed with the aid of the O'Brien periosteal stripper on the lower side; this maneuver facilitates complete costectomy at the time of the first posterior operation. In the anterior as well as the first posterior operation the intercostal muscle bundles are detached from the ribs with ease by this plan, because the separation is effected in the direction of the muscle

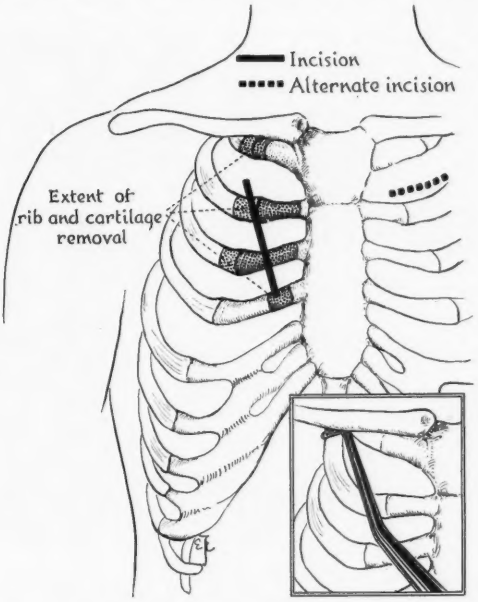


Fig. 1.—Anterior chondrocostectomy. A suggested plan of operation for thoracoplasty (O.H.W.). The incisions employed are indicated together with the extent of rib and cartilage removal. The oblique incision permits better exposure, but the operation can readily be done through a single transverse incision over the second rib. The resulting scar remains less prominent. The insert shows how the second and third ribs are freed from their beds and the intercostal muscle bundles attached to their lower margins.

insertion. Tearing and bleeding are in consequence minimized by this anatomic procedure. Inasmuch as the detachment of the rib from its bed is only partial (the superior border remaining attached) the viability of the rib is not threatened.

FIRST POSTERIOR OPERATION

The patient lies in the lateral decubitus, tilted slightly forward with the side to be operated on, up. Whereas from the standpoint of convenience to the operator the prone position is to be preferred, the comfort and minimal interference with ease of respiration dictate that the patient lie on his side. The skin incision is the usual "J" shaped one curving outward below the border of the scapula. After

division of the trapezius and rhomboideus, and latissimus dorsi muscles, the scapula is pulled laterally with a deep retractor. A jacket made like a harness (designed by Dr. Harry B. Hall of the surgical service) worn over the gown and shoulders by the second assistant and fastened onto the retractor with two snaps, facilitates retraction considerably. Through the exposure afforded by employment of this means of retraction, division of the fibers of the serratus anterior superior muscle becomes unnecessary. The insertion of the uppermost fibers of the serratus posterior superior muscle on to the second rib orients the surgeon with reference to the rib level. The third rib is removed first, being divided posteriorly. After retraction of the soft tissues and separation of the rib from its periosteum and posterior muscle attachments, the transverse process and rib can usually be cut together. Failing this, a satisfactory removal can be subsequently achieved with a rongeur. Employing the (right) O'Brien periosteal separator (left-sided thoracoplasty), the superior border of the rib can be disunited with ease throughout

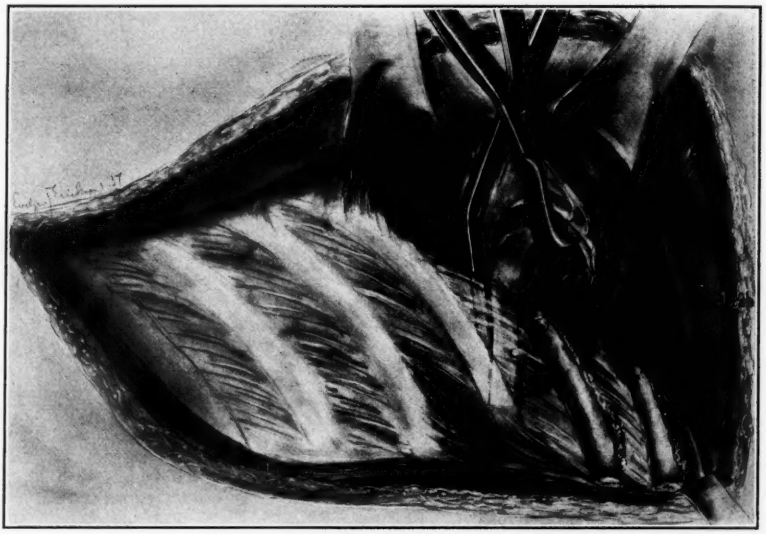


Fig. 2.—Drawing of upper stage posterior thoracoplasty illustrating the ease with which total costectomy is accomplished when anterior chondrocostectomy had previously been done, at which time the intercostal muscles were detached on the lower margins of the second and third ribs.

its entire extent. The separation of the lower border of the rib from its bed by the same means (left O'Brien), however, is not so easy owing to the direction of insertion of the intercostal muscle bundles. If the O'Brien separator (right) is pushed well posteriorly along the lower border of the second and third rib margins at the time of the anterior chondrocostectomy (as was described above), the procedure is very much simplified* (Fig. 2). The insertion of the intercostal muscle fibers in the first interspace to the lower margin of the first rib is such that the periosteal stripper pushed posteriorly at the time of the anterior chondrocostectomy

*Such a preliminary anterior section of ribs in operations for chronic empyema was described two years ago. (Wangensteen, O. H.: The pedicled muscle flap in the closure of persistent bronchopleural fistula. With description of preservation and employment of the intercostal muscle bundles by a process of ribboning [for the avoidance of abdominal hernia] in the obliteration of large chronic empyema cavities. J. THORACIC SURG. 5: 27, 1935.)

If at the same time the intercostal muscle bundles are detached along the lower border of the rib as here described, extensive rib resection for the obliteration of chronic empyema cavities should be considerably facilitated.

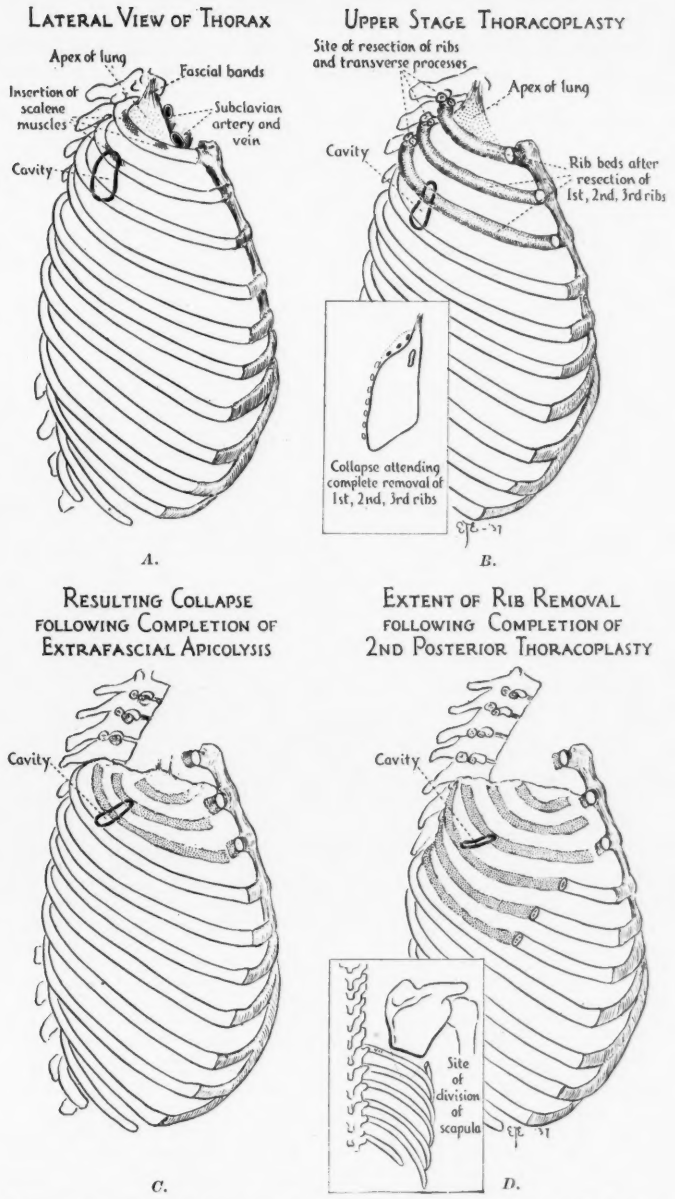


Fig. 3.—Diagrammatic sketches of lateral view of thorax to show effects of thoracoplasty and apicolysis on the lung.

A, Before operation. Note attachment of fascial bands.

B, After upper stage thoracoplasty there is collapse of the upper portion of the lung without descent of the apex.

C, After extrafascial apicolysis the apex has been immobilized and is now seen at a level of the fourth rib posteriorly. The collapse obtained in this manner approaches more closely to the ideal of concentric collapse and should permit more effectual closure of cavities.

D, After second posterior thoracoplasty. Resection of additional (two, three or four) ribs adds further to the collapse of the diseased portion of the lung.

does not readily separate the muscle from the rib. Previous anterior division of the first cartilage makes complete excision of the first rib a simple maneuver, however.

Since the publication of Carl Semb, of October, 1935, on extrafascial apicolysis came to our attention, this freeing of the pulmonary apex from the cervical neurovascular bundle has been done wherever feasible. In the main, the separation of the posterior aspect of the lung, permitting of visualization of the intramediastinal portion of the great vessels, has been accomplished usually with relative ease. Anteriorly, however, difficulty has more frequently been encountered. Fused solidifications between the lung, the endothoracic fascia and the periosteum of the first rib bed have made us somewhat hesitant over completing the apicolysis anteriorly lest a large pulmonary cavity be opened up. Yet, the increase in collapse of the pulmonary apex by this added procedure, even when incomplete, has served to augment materially the degree of anatomic collapse achieved by complete excision of the upper ribs. (Fig. 3.)

SECOND POSTERIOR OPERATION

A satisfactory anatomic result in our experience has not been accomplished by excision of the upper three ribs alone. In the usual second posterior operation, the segments from the fourth, fifth and sixth ribs are removed. The extent of this rib removal is indicated in Fig. 3*D*. When it is believed that removal of six ribs will afford a satisfactory collapse, the base of the scapula is frequently amputated in order to avoid catching the scapula on the seventh rib (Holman). The number of cases in which a greater vertical extent of rib resection has been deemed necessary is indicated in Table I.

TABLE I

DATA REGARDING THORACOPLASTIES, INCLUDING ANTERIOR CHONDROCOSTECTOMIES

A. NUMBER OF OPERATIONS PERFORMED, 146		B. NUMBER OF RIBS REMOVED	
Number of operations per patient	Number of patients	Number of ribs	Number of patients
1	1	3	2
2	2	5	2
3	18	6	9
4	19	7	8
5	1	9	6
6	1	10	12
		11	2
	42		41*

AUXILIARY PROCEDURES	NUMBER OF CASES
Extrafascial apicolysis (Semb)	10
Primary resection of transverse processes	7
Resection of scapula	4
Resection of cervical rib	1

*One patient died 22 days after partial excision of 4 ribs.

We have deliberately avoided being arbitrary concerning the extent of the performance of vertical thoracoplasty. Wherever the situation demanded more extensive removal of ribs in the vertical direction, it has been done without hesitation. The virtues and shortcomings of the partial thoracoplasty have been discussed at length by Holst, Semb and Frimann-Dahl.

POSTOPERATIVE CARE

Immediate Care.—The patients are returned to bed in the lateral decubitus with the operated side down. The foot of the bed is elevated routinely on a shock frame (Fig. 4). Such elevation serves to facilitate expectoration and at the same time aids

in supporting a satisfactory blood pressure. If the systolic pressure falls to 100 mm. of mercury or less, despite employment of this simple expedient, acacia solution is given intravenously.

Transfusions of blood are generously employed on indication in the preparation for as well as after operation. A hurried pulse (more than 120) in the absence of dyspnea constitutes indication for transfusion of blood.

Dyspnea.—When a patient complains subjectively of difficulty in breathing after thoracoplasty, oxygen administration is begun on little pretext. Despite complete excision of the upper ribs, severe dyspnea has rarely been observed. It was found necessary to administer oxygen postoperatively in only four of the forty-two patients mentioned in this report. This dreaded complication of dyspnea following the typical Wilms-Sauerbruch operation was an experience which very few surgeons escaped. In the type of procedure which we have done, it is rarely observed any-

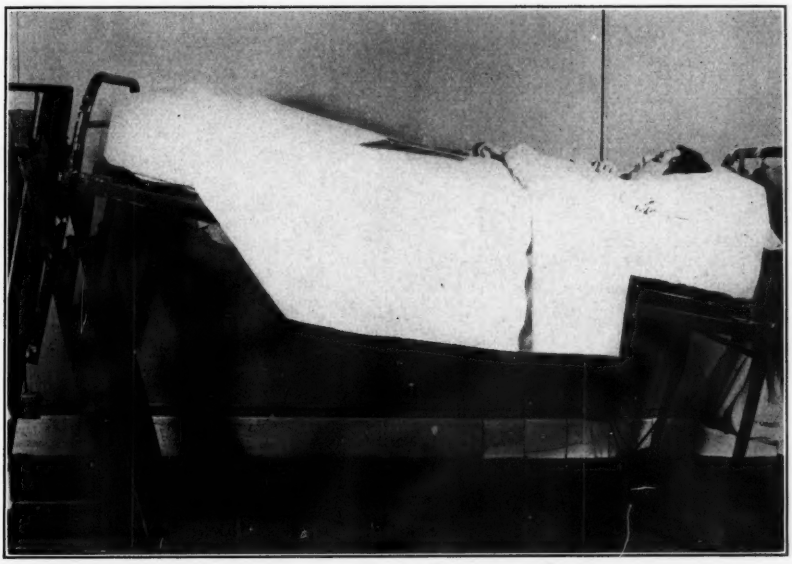


Fig. 4.—Frame used to elevate foot of bed in order to facilitate expectoration and support the blood pressure.

more. The striking absence of severe dyspnea attending the upper posterior operation is no doubt due in part to the limitations of rib resection to three ribs. Nevertheless, this number has been consistently removed in each upper posterior operation. We cannot escape the impression that the preliminary performance of the anterior costochondrectomy stiffens the mediastinal pleura on the operated side somewhat, decreasing naturally the possibility of an unstable mediastinum. In x-ray films made following the performance of the anterior procedure, we see not uncommonly unmistakable evidence of extensive layering of fluid over the upper portion of the corresponding lung field. Our patients represent a sanatorium group and are in no way unusual, and the commonly accepted criteria of indication for thoracoplasty have been applied to them.

When dyspnea or elevation of the pulse rate has occurred without apparent cause, a stomach distended with swallowed air has been suspected and has been carefully watched for by both staff and nurses. Such gastric dilatation is most likely to attend the second or third posterior thoracoplasty on the left side.

EMPLOYMENT OF EXTERNAL COMPRESSION

After the second posterior operation axillary compression with synthetic rubber sponges is commenced as soon as the wound is no longer tender, applied in the manner indicated in Fig. 5. Such compression is continued until osteogenesis has proceeded to the extent that the chest wall becomes firm (usually six to ten weeks after the last operation). It has not been our experience that excision of the anterior costal

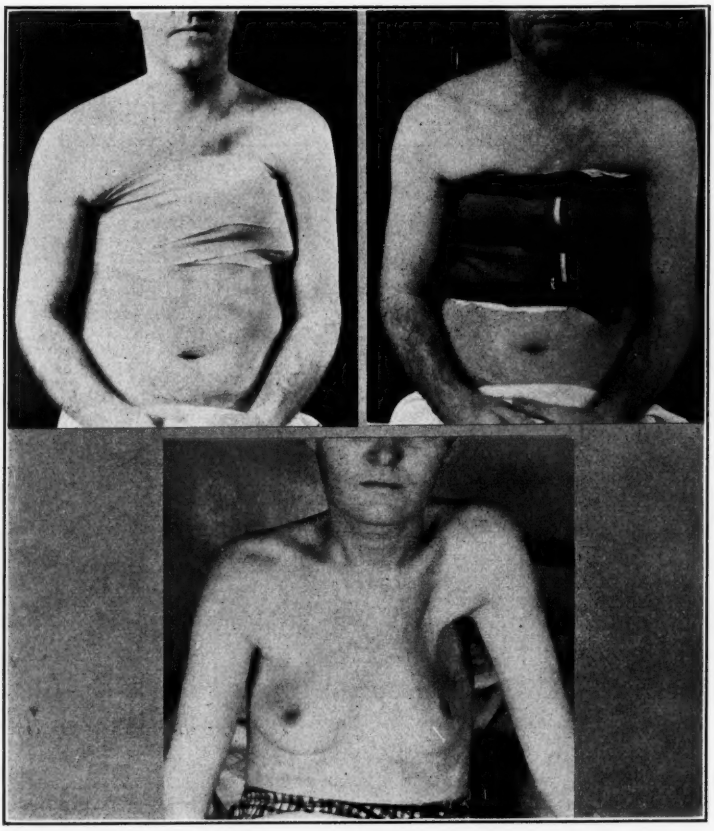


Fig. 5.—Methods and results of external compression.

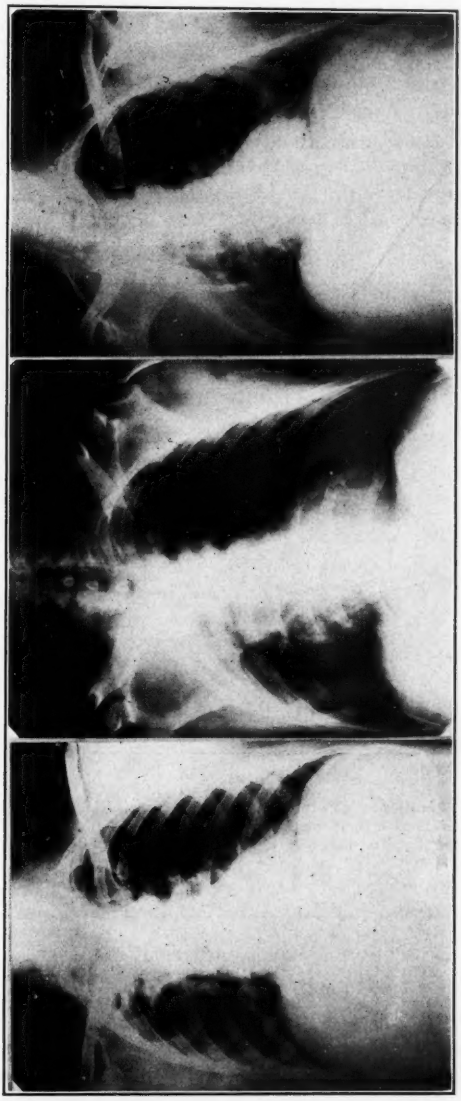
A, Synthetic rubber sponges are applied anteriorly and in the axilla. Compression is applied by means of an elastic bandage. This method may be applied a few days after operation when drainage from the wound has ceased and pain is diminishing.

B, A thoracoplasty belt has been found to be of value especially after the third posterior operation in patients who have had resections of nine to eleven ribs.

C, Illustrating degree of collapse of chest wall obtained after anterior chondrocostectomy, two stages of posterior thoracoplasty, and external compression. Note the elevation of the shoulder, and occasional accompaniment of the long posterior incision (see text).

cartilage contributes to the development of an anterior chest wall which is no longer firm or solid. We have yet to see an instance in which removal of cartilage resulted

in an infirm anterior wall. The degree of collapse which can be obtained with such external compression is illustrated in Figs. 5, 6, and 7. No routine attempt has been made yet to inhibit the regeneration of bone by treating the periosteum with chemical



A. *B.* *C.*

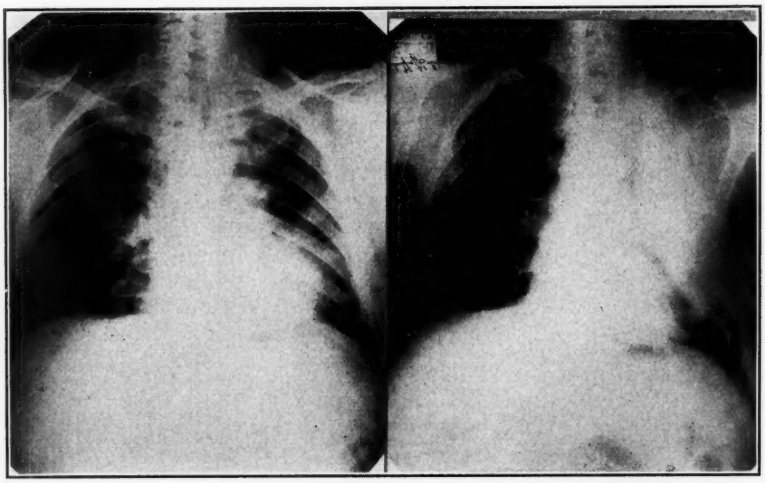
Fig. 6.—Films showing collapse obtained by thoracoplasty.
A, Before operation—cavity in right upper lobe.
B, After upper stage thoracoplasty. Note complete absence of upper three ribs.
C, Eight months after thoracoplasty (six ribs). Regeneration of ribs has occurred. There is excellent collapse and closure of cavity.

agents. On the contrary, it has been felt to be important to invite regeneration to insure a solid chest wall.

Whereas our patients have occupied the lateral decubitus position with the operated side down, up until the time that external compression is begun, it is doubtful whether

this is desirable. We have noted that many of our patients have, as a consequence of the operation and postural aftertreatment, an elevated shoulder on the operated side (Fig. 5*C*). Whereas this may be due in some measure to the operative trauma to the muscles, it is our impression that long continuance of the lateral decubitus position may have much to do with this undesirable complication. If maintenance of the lateral decubitus does not contribute materially to the collapse, this position should be abandoned early after each posterior operation. Certainly it is not a position of comfort for the patient.

No attempt should be made, however, to hurry the patient into subsequent stages. Full recovery from the first procedure should be awaited before the next operation is undertaken. The more quickly the two posterior operations can follow one another and the earlier that external axillary compression can be employed, the better will be the end result.



A. *B.*

Fig. 7.—Films showing collapse obtained by thoracoplasty.
A, Before operation. There is a large cavity in left upper lobe.
B, One year after thoracoplasty (six ribs). Collapse is excellent; cavity is closed; rib regeneration has occurred.

DATA CONCERNING PATIENTS AND PULMONARY LESIONS

It is impossible to present an adequate picture of the stage and extent of the pulmonary disease from which the patients suffered without giving detailed individual case summaries. Certain data, however, enable one to gain an impression of the general character of the lesions, and the condition of the patients. Of the 42 patients reported on here, the ages varied between 20 and 59 years, 12 being more than 40 years of age. Seventeen were males and 25 females.

Right-sided lesions predominated, the side of the major disease being the right side in 22 and the left in 16 instances. In 4 patients the in-

volvement was nearly equal in the two upper lobes. Altogether bilateral apparently active disease occurred in 20 (48 per cent). An attempt was made to determine from the x-ray films, the number of lobes involved with the result that only one lobe was found to be involved in 5 patients, two lobes in 23 patients, and three (or four) lobes in 14 patients. Measurements of the cavities as seen in the x-ray film revealed that 17 patients had cavities 2 cm. or less in diameter, 16 had cavities 2 to 5 cm. and 9 had cavities more than 5 cm. in diameter. Productive lesions predominated but 7 patients had considerable exudative tuberculosis. Atelectasis or extensive chronic lung fibrosis was present in more than one-third of the patients. Bronchiectasis was demonstrated by means of lipiodol in five patients, but as bronchography was not employed in a routine fashion the actual incidence of bronchiectasis was undoubtedly much higher.

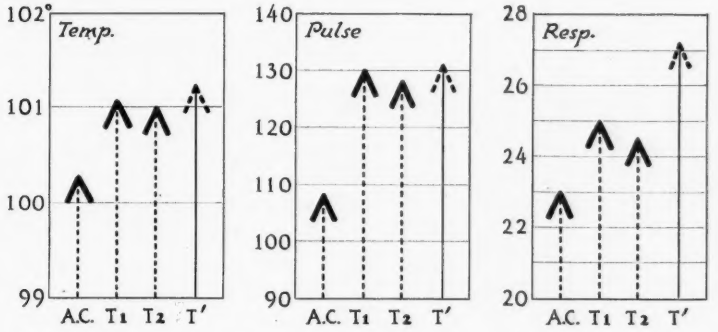


Fig. 8.—Average peaks of temperature, pulse rate and respiration rate after various stages of thoracoplasty (42 patients in each of the two series of cases represented).

Pneumothorax had been attempted at some time in practically all patients; 24 had previous operations on the phrenic nerve of the side concerned, 3 had hydropneumothorax, and 2 oleothorax on admission to the hospital. Patients with empyema were excluded from this series. Most of the phrenic nerve operations had been performed at an earlier date. Very few were done just preliminary to thoracoplasty, and during the past two years we have abandoned phrenicectomy immediately preceding thoracoplasty.

OPERATIONS

The extent of the surgery performed is indicated in Table I. The reactions of the patient to operations are indicated in Figs. 8 and 9 which record the maximum responses in temperature, pulse rate, respiration rate, and systolic blood pressure occurring after anterior chondrocostec-

tomy, first and second stage posterior thoracoplasty performed on the same 42 patients. Data are also included to permit comparison with the maximum reactions following upper stage thoracoplasty performed on the same number of patients prior to 1934 when it was the custom to operate on a greater number of ribs (usually four to six) but to resect shorter segments. In order to interpret the figures properly it is necessary to bear in mind that Fig. 8 dealing with temperature, pulse, and respiration responses refers only to maximum reactions, the peaks recorded being based upon averages of the individual peak reactions. The blood pressure figure (Fig. 9), on the other hand, merely records the number of patients in whom the lowest systolic blood pressure reading was above or below 90 mm. of mercury (and 80 mm. of mercury).

It is clearly revealed that the operation of anterior chondrocostectomy is much less formidable than posterior thoracoplasty. However, the

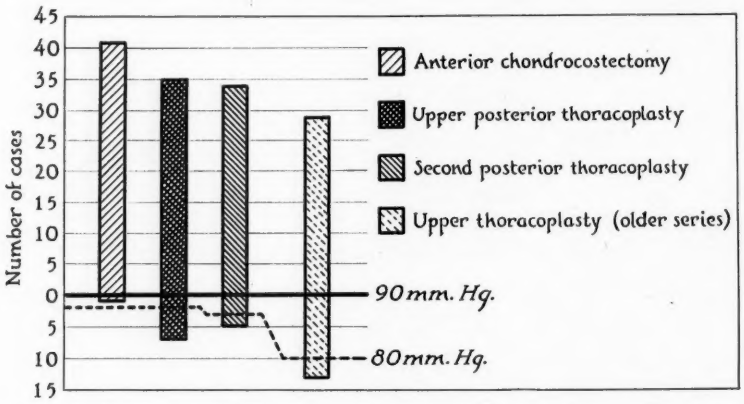


Fig. 9.—Number of patients whose minimum systolic blood pressure was greater or less than 90 mm. of mercury (and 80 mm. of mercury) after various stages of the thoracoplasty (42 patients in each of the two series of cases represented).

upper stage thoracoplasty is, as a rule, well tolerated. Comparisons with reactions to the earlier type of upper stage thoracoplasty show clearly that the total resection of the upper three ribs as now practiced is less traumatizing and safer than the earlier operation in which shorter segments were removed from a greater number of ribs. Dyspnea from a reduced vital capacity and a flapping chest wall, which we feared when the present plan of operation was first undertaken, seldom made its appearance.

The second posterior thoracoplasty, as Figs. 8 and 9 reveal, produces slightly less reaction, as a rule, than the upper stage operation. At this stage two, three or four additional rib segments of varying length are resected and the postoperative course varies greatly, depending on the extent of the resections and the ability of the patient to tolerate further surgery and further collapse.

When a third posterior operation is performed, shorter segments of ribs are resected than in the preceding stages. The postoperative reaction is usually correspondingly less, but with each succeeding stage the consequences of the operations are pyramided upon one another with the result that occasionally the reaction of the final stage is out of proportion to the magnitude of the operation.

COMPLICATIONS AND MORTALITY

The most serious complications encountered were the result of the aggravation of the pulmonary tuberculosis either by spread to the contralateral lung or by progression of a preexisting lesion. Five patients suffered such complications, and among them are numbered the two postoperative deaths in the series of 42 patients (146 operations). The case mortality, therefore, was 4.76 per cent and operation mortality, 1.37 per cent.

One of these deaths occurred twenty-one days after anterior chondrocostectomy, no further surgery being performed. The patient apparently should not have been operated on at this time as she had a recent history of recurring fever of low grade. The postoperative clinical diagnosis was tuberculous pneumonia; no necropsy was permitted.

The other death occurred in a poor risk patient who had bilateral disease. Death occurred eighteen days after the third posterior operation, one hundred and nine days after the upper stage thoracoplasty. Necropsy revealed only bilateral pulmonary tuberculosis. This patient had been recognized as a very bad risk from the beginning and had been accepted for surgery only after a long period of observation had demonstrated the futility of phrenic nerve crushing and bed rest.

TABLE II

IMMEDIATE RESULTS OF THORACOPLASTY

SERIES	NUMBER OF PATIENTS	COLLAPSE			CAVITY			CONTRALATERAL LUNG		DEATHS	MORTALITY
		GOOD	FAIR	UNSATISFACTORY	OPEN	CLOSED	DOUBTFUL	UNCHANGED	WORSE		
1934-1936 Chondrocostectomy	42	35	4	3	12	24	6	37	5	2	4.76
1931-1933	42	13	11	18	18	5	19	37	6	6	14.28

DEGREE OF PULMONARY COLLAPSE ATTAINED

The collapse of the thoracic cage resulting from the type of operation described here is strikingly greater than that resulting from the operations performed prior to 1934. The evaluation of immediate results is, of course, subject to individual interpretations, but based on a review of x-ray films taken within three weeks after completion of the thoraco-

plasties, the degree of collapse in our recent series was good in 35 patients, fair in 4 and unsatisfactory in 3, as compared with the earlier series in which collapse was good in only 13, fair in 11 and unsatisfactory in 18 (Table II). Among the earlier 42 patients operated upon after the manner of the Wilms-Sauerbruch operation between the years 1931 and 1933 there were six hospital deaths, or a mortality of 14.28 per cent, which was eventually the same as that noted in an earlier series reported in 1933 (Wangensteen) which overlaps the 1931-1933 series referred to here.

RESULTS OF THORACOPLASTIES*

It is still too early to attempt a final classification of results, but an effort has been made in Table III to evaluate the present status of patients observed for six months to three years after thoracoplasty. This preliminary survey indicates that 50 per cent of the patients have closed cavities and negative sputum. The two patients in group II who had bilateral apical cavities also have satisfactory collapse on the operated side, but will probably require bilateral collapse. Although some of these patients may lose ground in the future, others from group II and group V may eventually attain the favorable status of a negative sputum and closure of the cavity. Not much can be expected from those now classified as unimproved (group III), but improvement, even among these, is not impossible.

TABLE III

CLASSIFICATION OF RESULTS 6 MONTHS TO 3 YEARS AFTER THORACOPLASTY

		NUMBER OF CASES	PER CENT	WORKING
Group I.	Improved, sputum negative, cavity closed	21	50.0	7
Group II.	Improved, sputum positive or cavity open	9*	21.4	
Group III.	Unimproved	4	9.5	
Group IV.	Dead	3	7.1	
Group V.	Not followed (known to be alive)	5	11.9	

*Two patients have satisfactory collapse on operated side with cavity remaining in contralateral lung.

It will be noted that three deaths are listed. Reference has previously been made to two of these. The third death occurred more than a year after thoracoplasty and was independent of the pulmonary lesion, the patient having died of an acute exacerbation of chronic glomerulonephritis. This patient apparently had obtained satisfactory surgical collapse.

Whereas the addition of extrafascial apicolysis to the upper stage thoracoplasty augmented the anatomic collapse, there does not appear to be any evidence of improvement of the final result in the cases (10) in which Semb's extrafascial apicolysis was added to complete costectomy

*We wish to acknowledge here the helpful cooperation we have had from the superintendents of the State and County Tuberculosis Sanatoria in amassing the data. We are particularly indebted to Dr. H. A. Burns, Superintendent of the Minnesota State Sanatorium, for generous aid.

over the result achieved in those in which apicolysis was not done. Five patients among these have closed cavities and negative sputum. The others have not yet obtained satisfactory results. The failures in this group may be due to the fact that most of the cavities were large and that sufficient time has not elapsed to permit the patients to gain the maximum improvement. There does appear to be a tendency in some cases for the cavity to remain open—only sinking to a lower level with the greater compression of the upper lung by apicolysis.

The results we have obtained in our recent experience are not nearly so good as those reported by O'Brien in 1933 and 1934. He found that from 91 per cent to 96.9 per cent of the patients had closed cavities and 81 per cent to 84 per cent had negative sputum in different groups of patients. It is unlikely, however, that O'Brien's and our series of cases are strictly comparable.

The necessity of revision is one of the tests of the effectiveness of thoracoplasty although the decision as to the need of revision is a matter of opinion. Of the forty-two patients who had preliminary anterior chondrocostectomy, one has since had a revision and two others will require revisions if they are ever to have complete closure of their cavities. Two of these had extrafascial apicolysis in addition to thoracoplasty. By contrast, five patients of an earlier series of thoracoplasty required revision. In addition to these, there are undoubtedly other patients in both groups who would benefit by further surgery, but the number of instances of unsuccessful collapse is much greater in the earlier (1931 to 1933) series.

DISCUSSION

The need of improving the results of paravertebral thoracoplasty by secondary excision of portions of the costal cartilages was recognized as early as 1911 by Wilms. More recently a number of modifications of the operations of chondrectomy or anterolateral thoracoplasty have been proposed by Brunner, Bérard and Dumarest, Hedblom, Alexander, Haight and others, but in all these procedures the anterior stage was supplementary to posterior thoracoplasty. O'Brien has dispensed with the anterior operation by performing a total resection of the upper three ribs through a posterior incision.

Very little attention has been given to the possibilities of a preliminary anterior operation. Gale resected short parasternal segments of the costal cartilages before undertaking the posterior thoracoplasty in order to permit the remaining anterolateral rib stump to fall in, and in a paper published since our present method was adopted, Crimm, Strayer and Baker refer briefly to the employment of an anterolateral thoracoplasty as the initial stage. We have not found in the literature a description of a plan of procedure similar to the one presented here, nor have we found any discussion of the purposes or advantages of such a sequence.

The advantages of horizontal staging with the anterior chondrocostectomy performed first are as follows:

1. The anterior stage is the least formidable and provokes less postoperative reaction than other thoracoplasty procedures permitting early performance of the upper posterior operation.

2. The total removal of the rib segments through a posterior incision is greatly facilitated by the preliminary anterior resection and is well tolerated. There were no deaths following the upper stage thoracoplasty.

3. The scar tissue reaction provoked by anterior chondrocostectomy appears definitely to aid in stabilizing the mediastinum.

4. The employment of the sequence described here gives the maximum collapse obtainable by total resection of the upper ribs.

Unfortunately, the attainment of a perfect or ideal collapse of the thoracic cage does not always produce the desired improvement in the pulmonary lesion. There will always be failures due to underlying pathologic conditions such as thickened pleura, lung fibrosis, bronchiectasis, or very large or rigid-walled pulmonary cavities. There are undoubtedly patients whose condition is materially improved by thoracoplasty but who still remain invalids because of our inability to render them sputum-free by operation. Welles has recently described such a group of patients. Before attributing our failures to intrinsic causes, however, it is necessary to employ a technique that actually does produce the maximum collapse in the diseased portion of the lung. In our experience this maximum collapse is most readily obtained by the method described.

The question of whether extrafascial apicolysis further improves the final result has not been fully answered in our limited experience. This procedure appears to be theoretically sound. In a few cases brilliant results were achieved through its employment. In how many cases in a large group one may expect material improvement in the end results through the routine performance of extrafascial apicolysis, remains to be determined. Host prefers the extrapleural apicolysis to the extrafascial method of Semb and believes that it has a wider field of application. The suggestion recently made by Overholt that one excise at the second posterior operation the wall of tissue which forms in the bed of the first operation, would appear to be worth while.

SUMMARY

A new plan of procedure in the performance of thoracoplasty is here suggested in which an anterior chondrocostectomy is first done. Very little operative reaction usually attends its performance, permitting early execution of the upper stage thoracoplasty and therefore pyramiding the extent of the collapse attained by the posterior operation. In addition, complete costectomy of the upper ribs is considerably facilitated by the preliminary chondrocostectomy. This plan of operation in our

experience affords the maximum collapse and evokes the least operative reaction. Extrafascial apicolysis affords in suitable cases a valuable adjunct to the attainment of the best possible collapse. The employment of external compression in the postoperative period serves to augment the degree of collapse. An infirm anterior chest wall has not attended excision of the costal cartilages.

Thoracoplasty, because of decreased risk and lessened reaction, has now become available to a larger group of patients than formerly, but we cannot yet, with confidence, assure patients that thoracoplasty will achieve the degree of collapse afforded by a satisfactory pneumothorax.

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MANAGEMENT OF THE EXCESSIVELY MOBILE MEDIASTINUM IN THE SURGICAL COLLAPSE OF TUBERCULOUS PULMONARY CAVITIES*

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Secaucus, N. J.

THE possible unfavorable effects upon the mediastinum and the consequences thereof in operations on the chest, particularly when the pleural cavity is entered, have long been recognized and have been especially stressed since the advent of thoracoplasty and the more universal application of therapeutic pneumothorax. The subject has taken on even greater importance recently with the increased application of the procedures of lobectomy and pneumonectomy. In this paper the excessively mobile mediastinum is considered only as it affects the closure of tuberculous pulmonary cavities by artificial pneumothorax or thoracoplasty.

In our experience a shiftable mediastinum is encountered in most cases in which a pneumothorax is established and maintained for a variable period. The deviation is, as a rule, of little importance and does not interfere with the establishment of an effective collapse in those instances in which it is otherwise possible. There is, however, an ever present small group of patients in whom the mediastinum is so mobile as to serve as the primary obstructive influence to the attainment of a closed cavity. Thus in 600 consecutive cases in which pneumothorax was induced, artificial fixation of the mediastinum became necessary in 60, and in almost an equal number of other instances advantage was taken of a spontaneous pleuritis with effusion for the production of fixation.

The disadvantage of an excessively mobile mediastinum comes up for consideration again in thoracoplasty not so much as a factor in the interference with the achievement of an effective collapse, but more specifically as a causal mechanism of postoperative "spreads" to the contralateral lung and death from mediastinal flutter. In one series of 75 patients subjected to this operation during the past five years, 4 died of the latter complication and in 3 patients death was caused directly by a disseminated tuberculous bronchopneumonia of the uninvolved lung; in these 3, however, paradoxical movements of the mediastinum were a factor in the dissemination.

It is thus evident that the incidence of an unstable mediastinum is not so small as would first appear. Yet it is interesting to note that fixation

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of the mediastinum has been given an unimportant place in the extensive writings on oleothorax and that little can be found as to the method of induction and as to the type of case to be selected for the procedure. In this report, therefore, I intend to discuss fully the latter points and make brief reference to the mechanism by which a labile mediastinum interferes with the establishment of an effective collapse.

The experience of those versed in the technique of induced pneumothorax reveals that when the mediastinal structures are easily displaced in the presence of a partial pneumothorax, forcing the latter affects the pulmonary cavity little or none even when the intrapleural pressure is caused to become positive. All one achieves under such circumstances is either marked or complete deviation of these structures into the contralateral side or herniation of the mediastinum at one of its weak points. In addition to these mechanical effects, the patients exhibit dyspnea, tachycardia, and loss of weight. Effusion follows such a pneumothorax more frequently than is observed in otherwise partial pneumothorax, and subsequent malfixation of the mediastinum may take place.

These observations have led us to feel that the induction of a pleuritis with effusion for stiffening of the mediastinum has a definite place in collapse therapy. Once fixation is achieved, the mediastinum becomes a fixed point toward which the collapsed lung recedes by virtue of its attachments at the root. It then becomes evident that the layer of air in the pleural cavity can exert its influence directly on the lung. Even if pneumothorax should eventually fail to result in obliteration of the pulmonary cavity, the fixed mediastinum serves to lessen the risk of mediastinal flutter in those patients later subjected to thoracoplasty.

The following case vividly exemplifies the unfavorable mechanical effects of a labile mediastinum and equally convincingly demonstrates how its fixation in normal position may aid in bringing about closure of what appeared to be an uncollapsible cavity.

White female, aged eighteen years, whose family and past personal history have no essential bearing on the present illness other than contact with an open case of tuberculosis for two months prior to admission to the hospital. History of present trouble dates from February, 1934, and was characterized by a coryzal onset and the appearance of cough in March. A diagnosis of pulmonary tuberculosis was made in October of the same year, and the patient was admitted to our institution in December.

Admission findings were moderate fever marked loss of weight and positive sputum. The roentgenogram revealed a pneumonic cavernous process in the right upper lung field and a bronchopneumonic infiltrate in the rest of the lung. The left lung appeared relatively clear. Pneumothorax was immediately instituted, but, in spite of almost complete collapse of the lung and marked herniation of the anterior mediastinal space, the sputum remained bacillary and the x-ray picture showed residual cavity partly obscured by the shadows cast by the spine and sternum. The mediastinal structures were permitted to return to midline, and then fixation was attempted by the injection into the pneumothorax cavity of 10 c.c. of sterile mineral

Fig. 1. Fig. 2.

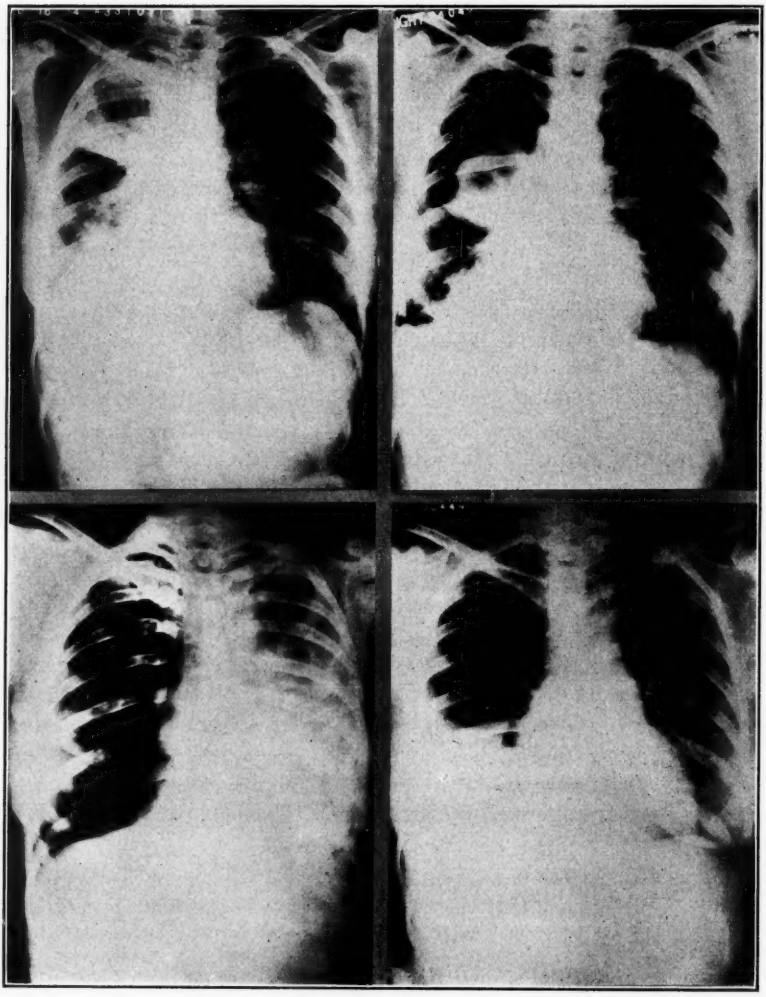

Fig. 3. Fig. 4.

Fig. 1.—Pneumonic cavernous process in the right upper half of the lung field. Bronchopneumonic infiltrate throughout the rest of the lung. Left lung relatively clear except for accentuation of the pulmonary markings in the midlung region.

Fig. 2.—Same patient after induction of pneumothorax. Note large cavity in pneumonic upper lobe.

Fig. 3.—Same patient after pneumothorax had been utilized to its optimum degree. Note almost complete collapse of the right lung with marked deviation of the mediastinal structures into the opposite side and a hernia of the anterior mediastinum as shown by the arrows.

Fig. 4.—Same patient after mediastinal structures had been returned to midline and a therapeutic effusion induced by oleothorax.

oil on July 3, 1935. This was followed by a moderate reaction and exudation into the pleural cavity. Fixation occurred within six weeks and closure of cavity was noted on the x-ray film of October, 1935 (Figs. 1 to 5).

The technique of fixation is vaguely described in Matson's[1] classical article on oleothorax. It seems that he used the procedure during the course of a compression oleothorax. A clearer discussion of the subject is to be found in the paper by McMahon.[2] The method about to be offered is one we have followed for the past five years and with which we have been very successful. It is relatively simple. The mediastinal structures are permitted to return to midposition either by temporarily discontinuing the pneumothorax or by daily aspirations of small quantities of air. When this has been accomplished, a liquid irritating medium

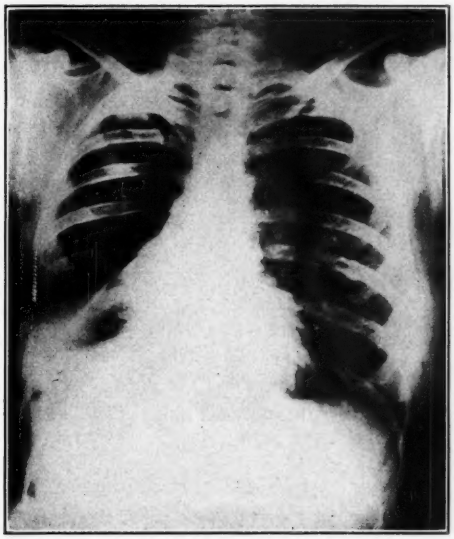

Fig. 5.—Same patient as shown in Figs. 1-4, after effusion had spontaneously resorbed and the mediastinum became fixed. Cavity is closed and sputum persistently negative.

is injected into the pleural cavity, and the patient is requested to lie on the sound side for an hour so as to permit more direct contact between the irritant and the mediastinal pleura.

Table I shows the various irritants being used by us. Maurer[3] uses 50 per cent glucose solution, and Rienhoff[4] produces a pleuritis with effusion for the purpose of stiffening the mediastinum by peptone broth.

TABLE I

IRRITANTS USED IN 60 CASES OF FIXATION OF THE MEDIASTINUM

NAME OF SOLUTION	QUANTITY	NO. OF CASES
Gomenol 2 or 5%	10-15 c.c.	35
Mineral oil	10-15 c.c.	16
Mineral oil followed by gomenol	10-15 c.c.	4
Hypertonic saline—7%	50-150 c.c.	5

While we had used gomenol almost exclusively in the beginning of our work, it is at present utilized only when mineral oil fails to provoke a pleuritis with effusion. We have found that mineral oil is less irritating than gomenol and that there comes with it a much lesser tendency to produce undesirable thickening of the pleura. The quantity of oil injected is 10 to 15 c.c. warmed to body temperature and previously sterilized by autoclaving for one-half hour at 15 pounds pressure.

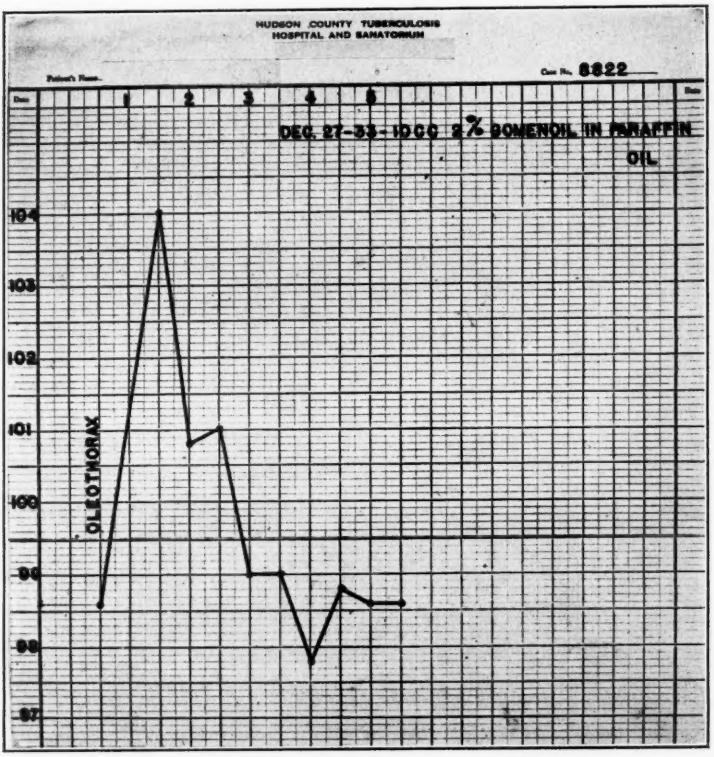

Fig. 6.—Showing temperature reaction to the instillation of 10 c.c. of gomenol into the pleural cavity. Note rapid return of temperature to normal within forty-eight hours.

The reaction to the injection of the oil follows from four to six hours later when the patient experiences pain in the chest and a feeling of warmth, often preceded by chills. The temperature may rise to 104° F. but in the average case does not exceed 103° F. Within twenty-four hours a small or moderate effusion is noted on fluoroscopy. The temperature usually subsides within seventy-two hours as is shown by the chart in Fig. 6, or it may take several days for it to do so as can be seen in Fig. 7. The symptoms enumerated as a rule disappear within the same period of time.

From the time of the reaction there follows a period lasting anywhere from four to eight weeks during which frequent fluoroscopic observations are made with three purposes in mind:

1. To note the behavior of the effusion and its effect on the mediastinum.
2. To maintain the pleural space.
3. To determine when fixation has taken place.

After the effusion appears, such observations become necessary in order to assure maintenance of the mediastinum in midposition. In the

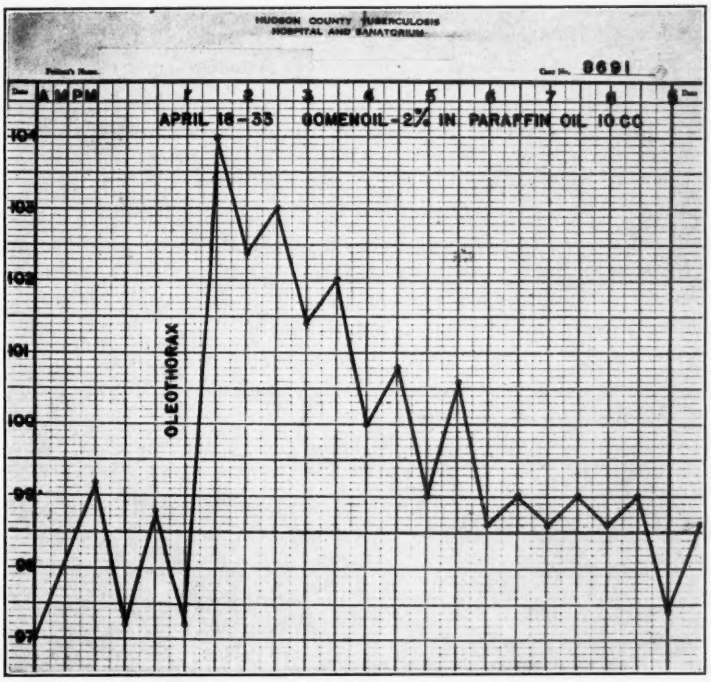

Fig. 7.—Showing temperature reaction to the instillation of gomenol into the pleural cavity and a much slower return of the temperature to normal.

vast majority of cases the fluid formed is not of sufficient quantity to displace the mediastinal structures, but if this occurs one must aspirate either air or fluid. In the 60 cases herein reported aspiration of the latter was resorted to in only 5 instances; in the remainder spontaneous resorption of the effusion took place within one to three weeks.

The next important step is the maintenance of the pleural space while one awaits completion of the stiffening process. Here again it has found that refills may not be necessary during this period, but as a rule small quantities of air (100 to 150 c.c.) have to be injected weekly to avoid union between the two layers of the pleura at any point. Caution

must be taken to avoid increase in the pneumothorax beyond the limit of keeping a thin layer of air about the lung in order that the mediastinum may not be displaced.

Finally, one has to determine when fixation is established so that the collapse of the lung may be increased in the attempt to achieve closure of the cavity. Fixation is considered complete when slight or no shift of the mediastinum is noted under fluoroscopic visualization on a deep expiration following a deep inspiration.

It happens occasionally that an effusion does not follow the injection of mineral oil intrapleurally, and the process must be repeated. The second instillation consists of 2 per cent gomenol; should this fail to cause fluid formation, as occurred in one instance, a 5 per cent gomenol solution is used.

The type of case to be selected for this procedure deepnds entirely upon the character of the existing pneumothorax prior to fixation. If the pleural space is limited in capacity by folds or wide bands of adhesions, it is best not to induce a therapeutic effusion because of the likelihood that encapsulation of the effusion and the tendency to rapid obliteration of the pleural space may follow. If cauterizable offending adhesions exist, pneumonolysis should precede fixation which may not become necessary if an effusion follows the operation. In summary, it may be stated that stiffening of the mediastinum is considered indicated only in those patients in whom the pneumothorax space is either entirely free of adhesions, or is traversed by unoffending cords or strings, or when the lung is suspended to the apical pleura but free along the rest of its surface.

Speaking of results, one must consider first the reliability of the method and second the effect on the pulmonary cavity. The answer to the first question is given in Table II. It is interesting to note that fixation, although not always complete, took place in every case. Even partial fixation, however, adds a sufficient resistant point in many cases so that closure of cavity eventually takes place by continuation of the pneumothorax.

TABLE II

TYPES OF FIXATION IN 60 CASES OF MOBILE MEDIASTINUM

Complete	46
Partial	14

While a stable mediastinum is desirable at a later date when thoracoplasty is considered in those cases in which pneumothorax fails to close the cavity, it serves a still more important function of immediate concern in that it spares some the operation and aids to turn the tide for the better in others in whom the cavity, previously resistant to closure, becomes obliterated. From Table III it will be seen that the collapse in this group of patients became effective in 50 per cent. This is a con-

siderably higher figure when compared with the results in a series of 398 cases of pneumothorax in which closure of cavity was obtained in 38 per cent. In 8 more patients various substitutions or supplementary procedures were added after the pneumothorax had been carried to its optimum degree, resulting in obliteration of the cavity in 7 patients.

TABLE III

EFFECT ON LESION IN 60 CASES OF FIXATION OF THE MEDIASTINUM

RESULT	PNEUMOTHORAX	PNEUMOTHORAX SUPPLEMENTED BY PHRENIC NERVE OPERATION	PNEUMONOLYSIS	THORACOPLASTY
Effective Collapse	30	2	2	3
Ineffective Collapse	22	0	1	0

Various questions may be raised regarding complications. It has already been stated that the effusion, including the oil used as the irritant, absorbs spontaneously in the vast majority of patients subjected to this procedure. In the 5 instances in which thoracentesis became necessary, the fluid presented the characteristics described by McMahon; namely, that of a clear or slightly turbid, greenish fluid in which tubercle bacilli were absent as revealed by careful culture and guinea pig studies. Subsequent observation of these patients for as long as five years proved not a single instance of empyema.

Another objection that may be raised concerns the resultant thickening of the pleura and the effect it may have on the capacity of the lung to reexpand at a later date, and on the tendency to premature obliteration of the pleural space. Thickening of the pleura is never more than moderate and seems to offer no more frequent obstacle to reinflation of the lung than follows spontaneous formation of an effusion during the course of artificial pneumothorax. As an illustration of this fact may be cited the 15 cases of this group in which the pneumothorax was voluntarily abandoned resulting in complete reexpansion of the lung in 14 and partial reinflation in one.

The tendency toward early obliteration of the pleural space following an effusion complicating a therapeutic pneumothorax is a factor always to be held in mind. Fortunately for the patient this complication does not occur frequently if both the effusion and the pneumothorax are properly managed, and especially if a constant pressure pneumothorax is maintained by the giving of frequent refills of small quantities of air until that tendency is overcome. Similar experience is encountered in the patients in whom an artificial effusion for fixation of the mediastinum is induced. Thus of the 60 patients comprising this study, indications of possible early loss of the pleural space were revealed fluoroscopically in 3, all of whom were managed successfully by the method just cited.

In conclusion I should like to stress that in properly selected cases fixation of an excessively mobile mediastinum increases materially the chances for the achievement of an effective pneumothorax. A stable mediastinum in an abandoned pneumothorax adds the further advantage of a decreased likelihood of postoperative mediastinal flutter in those subjected to thoracoplasty. For these reasons I believe that this procedure holds a definite place in a general collapse therapy program and that its use will probably be extended in the future.

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DISCUSSION OF SYMPOSIUM ON COLLAPSE THERAPY: "EXPERIENCE WITH COLLAPSE THERAPY FOR PULMONARY TUBERCULOSIS IN THE FIFTH AND SIXTH DECADES," BY H. R. DECKER; "PARTIAL THORACOPLASTY FOR PULMONARY TUBERCULOSIS," BY OWEN H. WANGENSTEEN, HERBERT A. CARLSON, AND WARNER F. BOWERS; "MANAGEMENT OF THE EXCESSIVELY MOBILE MEDIASTINUM IN THE SURGICAL COLLAPSE OF TUBERCULOUS PULMONARY CAVITIES," BY B. P. POTTER.

DR. HOWARD LILIENTHAL, New York.—Permit me to call attention to two points. One is the matter of compressing or, rather, encircling the chest with any kind of bandage. I think that it is a very serious thing for the patient. Even though there is only a small amount of energy required to breathe with the good side, it means a good many foot pounds of work in a day, and for that reason I have avoided in every possible way the encircling of the entire chest.

I can obtain compression of one side of the chest, the mobile side that has been operated upon, by a method which I have published several times, most recently in the *International Journal of Surgery,* with illustrations. I use elastikon, which I understand Dr. Alexander is also using. It was an invention of mine, given to Johnson & Johnson, who manufacture it. It has to be reenforced by another piece of plaster which we sew on at each end. It is not like the ordinary elastic bandage. It is tremendously powerful; you get genuine pressure with it. Unfortunately, it deteriorates in about a year, and the drugstores will not keep it, but you can obtain it from the manufacturers.

This is the back of the patient (illustrating on blackboard). Here is the plaster on the good side over the back of the shoulder. It goes around diagonally and posteriorly, then across anteriorly and takes its bearings from the iliac crest of the patient on the operated side. There is a tremendous pressure on that side. The well side is completely free, and the patient has no trouble whatever in breathing.

That is only one method. There are others, but they are more cumbersome.

I am not going into the details of operation, but I do want to bring up one more matter. Since I have been using the high frequency scalpel in these thoracoplastic operations, I have had much less trouble than I had before I used this instrument. It must be handled carefully, especially through the skin, and one must work quickly. If one works slowly with the electric scalpel, the edges of the skin become burned, and a good primary union will not result. Work in the deeper tissues does not demand speed.

My record is ten ribs without a single ligature, and perfect primary union (one case).

I am sure that if you try electric surgery in these cases you will be pleased. I use it in general operative cases, gall bladder, hernia, etc., but for thoracoplastic work, it is ideal. I have not employed it in intrathoracic work because of the danger of an accidental shock to the heart, which would be dangerous. I have heard that fatal accidents have occurred.

DR. HANS C. JACOBAEUS, Stockholm, Sweden.—In Sweden, we mostly do total thoracoplasty. We try wherever possible to do partial thoracoplasty. In the diseased parts of the lung we use a modification of Semb. In most cases we remove five ribs and usually have very good results. In my opinion there is one thing that is not so good; that is, when the lung is encapsulated in fibrous sheaths and it is impossible for it to develop again, and the other lung becomes infected. So one does an oleothorax when the other is complete without any tension at all. It is a greater risk, but I must agree that in many cases the oleothorax has given good results.

We have not employed plombage quite so much, but we have operated. After some years there are complications; the patients cough up much of the paraffin, so we have to operate on them again. If we do not obtain complete collapse immediately, we do not get it later, so we have quite some trouble with that method. We have seen very good results with small quantities of paraffin.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—Like Dr. Lilienthal, I am not going to discuss the technique of thoracoplasty in any way, although I should like to. I should like to comment on Dr. Decker's paper. With all due respect to Dr. Decker, I do not know that he has helped us very much except to point out the difficulties and the high mortality in this older group of patients; but the sad thing about the situation is that we are constantly (at least I am) having presented to us more and more patients in this age group.

Twenty years ago thoracoplasty was a new thing in this country, and it had to develop and be accepted by the physicians, and the physicians had to be educated to refer their patients to us for operation. The development has been very gradual. Each year thoracoplasty is becoming more accepted, and patients are being operated on earlier. We are getting more patients now to come in the early stages of the disease, but, also, along with them, many old patients who have had the disease a long time and have been struggling along with one form of treatment and another. They have had pneumothorax, which apparently was fairly successful at the time, have now been allowed to reexpand, and find that they are not cured by the pneumothorax, but their cavities have reopened. Instead of being 25 or 30 years old, they are 40, 45, or 50 years old, and they have cavity again or yet, and they want something done about it. What are we going to do? Thoracoplasty? But are we? I do not know.

I am uncertain as to whether there ought to be an age limit. As Dr. Decker pointed out, we cannot tell by examining a patient. When a patient 45 or 50 years old comes in, we can examine him carefully, listen to his heart, and make an electrocardiogram, etc., but we cannot tell whether he will survive thoracoplasty. The risk is certainly great. Of course Dr. Decker's figure of 8 deaths out of 17 thoracoplasties is appalling.

I should like to ask Dr. Decker a little more about the ages of those patients. He groups them 40 to 60 years of age, and possibly on one of his tables he said how many were 45 years of age, how many were 50, and how many were nearly 60. I did not see that on the table, but I should like to know if he can tell us very briefly whether those patients were mostly in their early 40's or whether most of them were in their late 50's.

Another great problem that is present in this country is the Veterans' Bureau. Many of us are connected with Veterans' Facilities. We are very apt to forget; we think of the World War as so present in our own lives, and such a recent thing that we do not remember that the World War was twenty years ago, and that every patient in a Veterans' Facility who served in the World War must necessarily now be more than 40 years old. We make plans for going ahead and treating these patients; we are cooperating with the Veterans' Bureau; and we are having round-table conferences with the Veterans' Bureaus at the various national tuberculosis meetings. We had one here several years ago. John Alexander made a plea to the Veterans' Bureau to cooperate with the surgeons to have thoracoplasties performed.

Each year that passes makes those men one year older, and if they have not had thoracoplasties yet, they will have to have them fairly soon, because all the veterans soon will be 50 years old. And are we going to do thoracoplasties on them then?

I have no suggestions to offer about this, but it is a serious problem. Dr. Decker has contributed something in considering this age group, and if anybody can give us any answers, or if anybody can find out how we are going to tell which ones to operate on, or how to operate we will be thankful. Naturally Dr. Decker's answer is (and mine would be, too), to operate less, to do smaller stages, and to try and ease them through. Even then we are going to have some difficulties.

It is a very difficult problem that we have to face in the next ten years.

DR. DEAN B. COLE, Richmond, Va.—I am convinced that the principle of a successful thoracoplasty is based on a process of whittling, rather than the master stroke of a standardized procedure.

Twelve years ago Dr. Johns began doing apical thoracoplasties for the closure of upper lobe cavities. For lack of a better name we called this "selective collapse" since at that time the accepted operation was to start at the bottom, cutting as much as possible of all ribs regardless of the size and location of the cavity. Then, as now, it was impossible to determine the number or location of adhesions keeping open a cavity; therefore it is impossible to determine beforehand in which direction a cavity will best collapse, or positively that the cavity can be collapsed.

We attempt to determine before operation the number of ribs and the length of sections necessary to remove in order to close the cavity. Should this first operation fail to produce the desired results, a second operation is planned and additional sections of ribs are removed depending upon the size, type, and location of the remaining cavity. This can be repeated if and when necessary.

A number of our patients who have had apical thoracoplasties were in the fourth and fifth decades. We have found that these patients will not tolerate abrupt changes in mediastinal pressure; therefore it is better to remove too short a section of rib and have to reoperate than to remove too much rib and lose the patient. In this connection, I wish again to stress the desirability of combining pneumothorax with apical thoracoplasty. We have not found it necessary or desirable to discontinue pneumothorax before doing thoracoplasty for closure of an apical cavity. Since pneumothorax pressure can be gradually and constantly increased, it is possible in this way to close apical or upper lobe cavities without the sacrifice of too much rib, lung or patient. With this procedure intrapleural pressure is controlled by the insertion of a pneumothorax needle at the time of operation.

I was glad to hear Dr. Potter's discussion on fixation of the mediastinum. We have been doing this for a number of years and our experience is not dissimilar to his.

In summing up, I feel that we should plan the procedure, depending on the size, the location, the type of cavity, thickness of the wall of the cavity, and the presence

and location of adhesions. We cannot determine accurately all these factors, but we can predict a number of them. If we study our patients individually, I think we shall have better results.

DR. HOLLIS E. JOHNSON, Nashville, Tenn.—I wish to discuss Dr. Decker's paper by relating the experience that Dr. Blalock and I have had with four patients, 58 years of age and over. Two of these were males and two were females. Three of the patients had pneumothorax, and three of them had thoracoplasties; that is, two of those who had pneumothorax had to have thoracoplasty. In none of them was the pneumothorax collapse satisfactory, and in all it had to be abandoned.

One of them was a physician 58 years old with bilateral tuberculosis and a huge cavity involving practically all the upper lobe of the right lung. We attempted pneumothorax and obtained a collapse which was not satisfactory. Dr. Blalock did a thoracoplasty on him and he made a very satisfactory recovery. He is now 62 years old and has been in the active practice of medicine for more than two years, having a country practice. He had seven ribs removed in two stages.

Another, a white woman 59 years of age, had a thoracoplasty and now, five years later, she is still alive and apparently doing quite well.

Another white woman, also 59 years of age, had a pneumothorax because of a unilateral tuberculosis and a rather large cavity about the diameter of a silver dollar. The cavity could not be closed by pneumothorax and Dr. Blalock did a thoracoplasty. She, also, made an uneventful recovery and is alive today, almost six years after the thoracoplasty. She had seven ribs removed in two stages.

Another patient, a male 62 years old, had unilateral pulmonary tuberculosis with a cavity. I attempted pneumothorax. The collapse was not satisfactory, and the patient developed evidences of circulatory failure. For this reason we abandoned the pneumothorax and did not attempt to do a thoracoplasty, or any other form of collapse. The lung reexpanded without leaving the patient any worse because of this experience.

These four cases without a fatality prove nothing at all because of the small number; but we certainly are encouraged, because of the results obtained in these patients, to continue collapse in this age group whenever the occasion arises.

DR. EDWARD N. PACKARD, Saranac Lake, N. Y.—I should like to ask Dr. Potter how long he waits before he puts oil into the pleural cavity in order to stiffen the mediastinum. In my experience, where a mediastinal shift has occurred, or a mediastinal hernia is present, by increasing the interval between refills or by limiting the amount of air injected, the mediastinum will come back into place, or the hernia will disappear so that the pneumothorax can be continued. This procedure may take several weeks, but I have never seen a mediastinum that, when once allowed to come back into normal position, did not stay there. So I have not used oil to produce a pleuritis, and I wonder whether the disadvantages or the dangers of the use of oil are not greater than that of allowing the mediastinum to swing back, even if we do not get the pulmonary compression as quickly as we would desire.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—I should like to speak a word in elaboration of the presentation by Dr. Carlson. He and I have been very much gratified with the lesser operative reaction which has attended employment of this procedure. Successes are to be weighed not only in the light of the operative risk and the improvement which accompanies employment of a certain procedure, but the nature of the operative reaction often affords a better clue concerning the risk assumed by the surgeon for his patient than does the mortality table. It is difficult to know whether a procedure which one or a number of surgeons employs is the best

or most appropriate. There are no critical criteria other than those of the general nature just mentioned which help to appraise properly the value of a given operation. Most surgeons eventually learn from their own experience which procedure to apply with greatest safety and benefit to their patients.

I like to look upon an operation as a general looks upon a campaign of battle. To be sure, there are many kinds of generals. There are those who insist upon winning the battle as quickly as possible—no matter what the cost. Some generals hold to this belief as the best course of action. Others are hurried into this impatience knowing how often governments change generals because of anxiety for early victory. There would appear, however, to be no justification for surgeons to be so motivated. What every sound and safe surgeon should insist upon for his patient is the greatest gain at the least cost. Time is a secondary consideration. Strategy should be a primary consideration among surgeons when they gird themselves to do battle for their patients.

The thoracoplastic operation which we have been doing is an anatomic procedure. The preliminary anterior chondrocostectomy facilitates rib removal at the time of the first stage posterior thoracoplasty and minimizes the operative reaction incident to it. In our experience, firm anterior chest walls have uniformly followed excision of the upper costal cartilages.

There is an important item in the procedure which Dr. Carlson passed over somewhat lightly. The nature of the attachment of the intercostal muscle bundles to the ribs is such that the upper border of the rib can be completely separated from its periosteal bed when the periosteal separator is pushed forward from behind; with reference to the lower border of the rib, the separation is best done in the other direction. Advantage has been taken of this anatomic peculiarity of insertion of the intercostal muscle bundles. At the time of the anterior chondrocostectomy, the periosteal separator (the O'Brien instrument or a variant of it) is pushed along the lower margin of the rib, and with surprising ease the periosteum is readily separated from the rib as far back as the angle of the rib. This maneuver is carried out on both the second and third ribs; the intercostal muscle bundle cannot be separated completely from the first rib in this manner. Detachment of half of the periosteal bed does not vitiate the viability of the rib. When the upper thoracoplasty is done and second and third ribs are sectioned near the spine, the entire rib can then be withdrawn after the periosteum has been detached from the superior border.

I have applied this principle to the second stage operation too, dividing the fourth, fifth, and sixth ribs through a short axillary incision, at the same time freeing the ribs from their periosteal beds on their lower surface. A week later, the second posterior operation can be done through a small incision from behind. It will always, of course, be necessary to expose the greater extent of the first rib, but I am inclined to believe that the employment of this maneuver in rib removal may lead to considerable simplification of the operation of thoracoplasty.

Similarly in chronic empyema, extensive rib removal may be accomplished with this plan of operation through very short anterior and posterior incisions. Rib removal by this process of tunneling is, as I have previously indicated (J. OF THORACIC SURG. 5: 27, 1935), more readily accomplished in chronic empyema than in thoracoplasty for tuberculosis; for in empyema, the ribs are often loosely attached to their rib beds.

In this Association, for years we have heard our modern St. Patrick and Homer struggling to compose their differences over many problems of thoracoplasty, and we who listen are frequently left with the impression that the width of the gap is more often widened than bridged by such discussion. Each continues to per-

form his procedure in the manner and on the indications which experience has taught him to be best. The sequence of operation which Dr. Carlson described and the manner of rib removal which we have employed constitute in our experience a satisfactory manner of attaining the maximum of collapse with minimal trauma and we recommend it for your favorable consideration.

There are a few other matters of which I should like to speak briefly. With reference to Dr. Lilienthal's comment concerning the employment of external compression, may I say that I like to find myself in complete agreement with my superiors, and in the presence of Dr. Lilienthal, I bow my head in deep respect. Yet, I must say that we have employed external compression much in the manner described by Dr. Carlson since 1932, when I had the opportunity of seeing how effectively Dr. John Alexander employed external compression after thoracoplasty. In this connection, it is well to remember the admonition of John Hunter to his friend, Eduard Jenner, "Don't think; try the experiment." External compression, as we have employed it, serves to augment the collapse obtained by the surgeon and is borne by the patient without complaint.

The paper by Dr. Hart prompts me to say a word about wound healing. Dr. Hart, who was trained in a school where he observed the frequent use of silk, spoke this morning concerning the use of catgut for ligatures and sutures in thoracoplasty. I was reared in a school of catgut and find myself more and more enthusiastic over the employment of silk. My dissatisfaction with catgut arose over the frequent rupture of long vertical abdominal incisions, especially after gastric resection for cancer. Since I began closing such wounds (both peritoneum and fascia) with interrupted silk, not a single evisceration has occurred in our clinic. Now every vertical incision in the abdomen is closed with buried silk. Extrusion of ligatures has been a most unusual occurrence. Silk is an inert substance, whereas catgut, as Harvey and Howes have shown, invites a much greater cellular reaction.

Dr. Carlson and I have had some misgivings over the extrafascial apicolysis. Yet we leave the operation with a greater sense of satisfaction in having accomplished a good anatomic collapse than when we omit the procedure. We have been dismayed now and then to see that the cavity still persists open, having only sunk to a lower level in the thorax. Yet, I do believe that Semb's extrafascial apicolysis adds something worth while to the operation of thoracoplasty.

DR. H. R. DECKER, Pittsburgh, Pa.—It should be understood, I think, that in the group of cases which I reported upon this morning, all of them were individuals who were incapacitated and who had at different times tried treatment without success prior to collapse therapy. Economically, they were a drug on the market, either domestically or institutionally, and from any standpoint that we could see them, none of them, it seemed to us, was likely to change his status by any future care unless collapse therapy was undertaken.

I think a situation of that sort always tempers one's judgment as to what one will advise. Some individuals choose to live a longer period of time, even though they are invalids, rather than take the risk of having their lives shortened by operation.

I think the only hazard of collapse therapy in this old-age group is thoracoplasty. I am perfectly appreciative, as Dr. Welles pointed out, that the result in this very small series of 17 cases was not a very happy one. Yet, when the group is analyzed, it is found that 2 of the 8 patients who died were past 50 years of age, one 50 years old and one 54; 4 of them were between 45 and 50, and the other 2 between 40 and 43 years of age.

Two of those patients did not die as a result of the operation. One of them we considered arrested, and the other one we considered stationary, likely to die of the disease and not helped by the operation.

The operation, it seemed to us, was directly contributory to the mortality in only 4 of the patients. In only 2 was there what might be called a definite operative death, that is, within forty-eight hours, one of those cases resulting from a rupture of the subclavian artery, and the second one developing a circulatory syndrome that I described in the paper.

I feel that the results of other members of this Society and from other sources have not been published fully enough, that it is a topic of great importance, and we shall learn if other surgeons will report their records.

Dr. Johnson is to be congratulated on the results that he obtained in his cases. We have tried to establish for ourselves contraindications in this age group for thoracoplasty. On the basis of our experience, I should say that there are three or four very definitely. In the first place, if there is any suggestion at all of nephritis, those patients should not have thoracoplastic procedure; in the second place, if there is any suggestion of cardiac symptomatology, either subjective or objective, they should not have a thoracoplasty. In the third place, if their vital capacity is less than 1,000 c.c. by successive tests, they should not have thoracoplasty.

If there is any suggestion of extreme fibrosis of the other lung with accompanying emphysema, the patient should not be given thoracoplasty.

DR. HERBERT A. CARLSON, Minneapolis, Minn.—Regarding this question of external compression, we have had difficulty in getting the compression exactly where we wanted it; namely, high in the axilla and under the scapula. I have not tried the method that Dr. Lilienthal suggests, but I should like to try it. However, I am inclined to believe that it will not give us the compression as high as we want it. The method we have been using with the circular compression has not done any harm so far as I can see. Sometimes we have not been able to apply the external compression as tightly as we at first attempted. The patients complained of pain rather than dyspnea. Even with the method that we have used we have not reached the ideal. We are still looking for a better method for applying external compression.

Much has been said about the age of the patients in this discussion. I want to say of our patients that 12 of the 42 were more than 40 years of age, the oldest being 59 years. There were no deaths in the series of the older patients.

DR. B. P. POTTER, Secaucus, N. J.—I failed to read parts of my paper for two reasons: In the first place, I was very conscious of Dr. Meade's communication instructing me to keep the paper within fifteen minutes, and in the second place, this being my first appearance before this association (although I have been attending its meetings for the past five years) and knowing the critical way that the members consider each presentation, I was a little nervous in anticipation of what may come.

With the remarks of Dr. Jacobaeus, I am in perfect agreement. We have used oleothorax for the past six years, and have come to feel that its use should be limited primarily for fixation of the mediastinum and occasionally to maintain an effective pneumothorax when obliteration of the pleural space is threatened. We have discontinued its use in empyemas. Its application in stiffening of the mediastinum is only incidental, for other media than oil may be used. Thus, Moore used 50 per cent glucose and Rienhoff uses peptone broth.

The effusion, as well as the oil, absorbed completely in 55 of the 60 cases. This incidence of spontaneous resorption is certainly higher than is seen to occur in the effusions following induced pneumothorax.

Dr. Cole's statement with reference to the sticking out of the lung to the lower chest wall, is well taken. I think it is a desirable feature when a maintenance oleothorax is considered and under certain conditions in bilateral pneumothorax

therapy. I prefer, however, not to have the lung stick out in other instances, because once there is a point of attachment, obliteration of the remainder of the pleural space may form very rapidly.

In answering Dr. Packard's question, I would say that bringing the mediastinum back to midline and holding it there for a variable period of time may and does lead to fixation when the mediastinum presents slight or moderate mobility. If, however, the movements are excessive, fixation does not take place by this method—at least not in our experience. The interval between induction of pneumothorax and fixation of the mediastinum varies. On the whole, I would say that anywhere from several weeks to several months may pass before fixation is attempted. Certainly one should not wait longer than six months for we have found in 600 patients subjected to pneumothorax in the past six years that, if an effective collapse is going to take place at all, it occurs within a period of six to eight months. After that, pneumothorax seldom becomes effective and must be abandoned for some other form of surgery.

As I have already pointed out, nothing new has been offered except a definite method by which fixation may take place, which heretofore had not been described. That a fixed mediastinum is desirable, and in fact often essential, is evident from the remarks of Dr. Rienhoff in his paper and from the references to this subject repeatedly made by various speakers during this session.

THE EFFECT OF PHRENIC NERVE INTERRUPTION ON THE GASTROINTESTINAL TRACT*

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ATTENTION has been called to the changes occurring in the shape and position of the stomach following phrenic nerve exeresis by Mattina, Rickers, Lichtenstein and others. Mention has also been made of the gastrointestinal disturbances which result from phrenic paralysis. Most of the emphasis has been placed on the gastrointestinal symptoms which follow left-sided exeresis, although cases have been reported describing gastrointestinal symptoms resulting from right-sided exeresis. In this study the chain of events taking place in the gastrointestinal tract following paralysis of either the left or the right phrenic nerve is to be considered separately.

Maendl and Schwarzmann reported their experiences in 100 phrenicectomies. When the left side of the diaphragm was paralyzed, almost all their patients suffered from loss of appetite, a feeling of fullness, and vomiting immediately after the operation. Welles reported 300 phrenicectomies and found that in a small number of cases the rise of the diaphragm was followed by digestive upsets. The majority of these upsets were temporary and were more common following left-sided than following right-sided phrenicectomy.

Stanbury reported 11 autopsies on cases in which previous phrenic paralysis had been produced. In 10 of the 11 cases there was marked distortion of the abdominal viscera, in three of these cases there was gastroduodenal obstruction. Thomas and I, in a previous communication, reported 4 cases in which acute dilatation of the stomach followed thoracoplasty performed subsequent to left-sided phrenic paralysis. Ballon, Wilson, Singer and Graham, in an experimental study, found that when left-sided phrenicectomy was followed by a marked rise of the left side of the diaphragm, there was an increased angulation of the abdominal portion of the esophagus. They could find no experimental evidence, however, to indicate that phrenicectomy results in obstruction of the lower part of the esophagus or proof that phrenicectomy may produce cardiospasm. Francis Trudeau noted a number of cases in which gastric disturbances followed left-sided

*From the Southern Pacific Sanatorium, Tucson, Arizona.
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phrenicectomy. While he found that these disturbances might last for a week or even several months, he states that they always eventually disappear.

In this study 173 cases of phrenic paralysis have been reviewed. There are three points which seem to be particularly significant. First, gastrointestinal symptoms may follow phrenic interruption on either the left or the right side. There seems to be a syndrome following left-sided phrenic interruption, which is characteristic and differs from the syndrome following right-sided phrenic interruption. Second, anatomic changes can be demonstrated in the gastrointestinal roentgenograms taken before and after phrenic operations, which account for the observed symptoms. Third, the gastrointestinal symptoms following temporary phrenic nerve interruption have a tendency to be transient, whereas the symptoms following permanent phrenic exeresis may persist for a number of years, and definite pathologic changes may be produced in the gastrointestinal tract.

All the patients in this study were from the Southern Pacific Sanatorium in Tucson, Arizona. The patients were almost all adult males. Most of them were hospitalized for a period of one year, during which time they received the same attention and followed the same routine and diet. It is therefore unlikely that dietary indiscretions were a factor in causing the gastrointestinal symptoms. Also, alcoholic beverages and tobacco were prohibited.

After becoming interested in this subject, I personally interviewed 44 patients before and after the phrenic operation. Thirty-three, or 75 per cent, of the patients interviewed gave a definite history of gastrointestinal disturbances which could be attributed directly to the operation. I also reviewed the records of 129 additional cases. Twenty-two, or 17 per cent, of the 129 cases had gastrointestinal symptoms of sufficient severity to necessitate consultation with a gastroenterologist. In all of these 22 cases gastrointestinal or gall bladder roentgenograms were made.

It was clearly demonstrated that paralysis of the left or the right hemidiaphragm may result in a chain of symptoms which is characteristic of the left or the right side, respectively. The syndrome following paralysis of the left hemidiaphragm is characterized by decreased appetite, feeling of fullness often associated with nausea and vomiting. There is a notable absence of pain. On the other hand, the syndrome following paralysis of the right hemidiaphragm is characterized by pain in the right upper quadrant and a chain of symptoms suggestive of gall bladder disease. In some of the right-sided cases there were also decrease of appetite, distention, and nausea. The pain in many of the patients with right-sided phrenic interruption was so

definite and suggestive of gall bladder disease that in 3 cases cholecystectomy was done unnecessarily. In 3 additional cases appendectomy was performed without relief (Table I).

TABLE I

COMMONEST SYMPTOMS FOLLOWING PHRENIC NERVE PARALYSIS

SIDE	DECREASED APPETITE	GAS OR FEELING OF FULLNESS	NAUSEA OR VOMITING	PAIN	ABDOMINAL OPERATIONS FOR SYMPTOMS
Left 22 cases	20 (91%)	15 (68%)	10 (45%)	1 (5%)	0 (0%)
Right 29 cases	13 (45%)	9 (31%)	5 (17%)	27 (93%)	6 (21%)

The symptoms in the 44 patients who were personally interviewed were classified according to their severity. In 12 of these cases the symptoms were considered severe because they were definitely impairing the patient's progress. In 10 of the patients the symptoms were considered moderate because they were causing discomfort sufficient to be brought to the attention of the attendants, although the patient's progress was not being impaired. In 11 cases the symptoms were classified as mild because their presence was discovered only after careful questioning. Ten of the patients had no symptoms. The rise of the diaphragm as measured in centimeters was not in direct proportion to the symptoms. The presence of pneumothorax had no effect on these symptoms. In the 22 cases of gastrointestinal disturbances, which were taken from the records, the symptoms were of necessity severe or moderate in order to demand sufficient attention to be recorded on the history (Table II).

TABLE II

EVALUATION OF SYMPTOMS FOLLOWING PHRENIC NERVE PARALYSIS

DEGREE OF SYMPTOMS	NUMBER OF CASES	AVERAGE RISE OF DIAPHRAGM	PHRENIC COMBINED WITH PNEUMOTHORAX
Severe	12	6 cm.	2
Moderate	10	5½ cm.	4
Mild	11	3½ cm.	3
None	10	5½ cm.	3

There were 22 cases from the records. In all these cases the symptoms were severe or moderate. The average rise of the diaphragm was 4½ cm.

The onset of the gastrointestinal symptoms appeared immediately after the phrenic operation in 27 cases. In 28 cases the onset was delayed from two weeks to as long as three months. In 2 patients, one month after a temporary phrenic nerve operation, there were only slight roentgenographic changes in the stomach and no symptoms. Three months later, marked roentgenographic changes in the stomach had appeared, as well as definite symptoms.

The anatomic and pathologic changes which may occur following paralysis of the left or right hemidiaphragm are best described by Stanbury, who made his study from 11 autopsies. I shall quote directly.

"In both cases of left-sided phrenic paralysis, the stomach occupied a vertical position in the upper left quadrant. The cardiac portion was displaced upward and was dilated. The greater curvature was directed forward. The duodenum was displaced to the left. The small intestine lay in the pelvis. The transverse colon was not displaced upward in either case. In one case the stomach was tremendously dilated. In its upper two-thirds, the greater curvature pointed laterally, and in its lower third, anteriorly, producing a constriction across the anterior surface of the stomach. Both loculi so formed were ballooned out and tense. The duodenum was markedly dilated for a distance of 7 cm. beyond the pylorus. On opening the stomach it was found to contain some blackish foul-smelling fluid. The stomach wall was thinned out and the rugae had disappeared. The duodenum contained the same type of fluid material.

"In 8 cases of right-sided phrenic paralysis showing visceral deformities, the longitudinal axis of the stomach tended to lie transversely. The organ was elongated and shifted to the right to a varying degree. The fundus faced downward and the pylorus was elevated so that it lay along the margin of the gall bladder. Two of the stomachs presented a uniform constriction in their midportions, giving the organ an hour-glass appearance. In three cases the stomach was tremendously dilated. The gastric wall was thin and the rugae were ironed out. The duodenum was very much dilated and boggy. The superior mesenteric artery was pulled tightly over the duodenal-jejunal junction and the bowel distal to it was collapsed. In one case the whole mucous surface of the dilated duodenum was congested and weeping blood. There was no evidence of ulceration. In each of these cases the small intestine was down in the pelvis."

In the present study, roentgenograms of the stomach and gall bladder were made before and after phrenic interruption in 12 unselected cases. The findings in the roentgenograms correspond with Stanbury's findings from his autopsy material. After interrupting the left phrenic nerve, the pylorus of the stomach moved to the left from 2 to 6 cm. and down an average of about 2 cm. The fundus was raised and greatly distended with gas. The stomach was suspended perpendicularly downward, and the pylorus was bent at an acute angle (Fig. 1), so that the pyloric portion formed a separate loculation.

After interruption of the right phrenic nerve, the pyloric end of the stomach moved 2 or 3 cm. to the right and 4 or 5 cm. higher. The stomach lay in a transverse position with the fundus definitely nar-

rowed, and the normal gas bubble absent. In most cases there was a constriction at the midportion of the stomach causing a definite hourglass appearance (Figs. 2 and 3).

The mechanism involved in the changes described is best understood by fluoroscopic observation of the stomach. The normal stomach moves upward on expiration and downward on inspiration. This change in position averages about 2 cm. at the pylorus and 4 to 6 cm. at the fundus.

After paralysis of the left phrenic nerve, fluoroscopic examination shows the fundus of the stomach to be stretched and stationary, while the pylorus moves with each respiration. Joannides has described the sphincter action of the diaphragm on the esophagus and has shown that there is an obstruction in the region of the cardia coincident with each inspiration, and a relaxation with release of the obstruction with each expiration. Acute dilation of the stomach, therefore, may result from paralysis of the diaphragm. This probably explains the presence of the large gas bubble found in the stomach after paralysis of left phrenic nerve.

When the right diaphragm is paralyzed, the right half of the stomach becomes fixed, while the left half moves with each respiration, thus causing the apparent hourglass deformity. Mattina found in his case that after paralysis of the right phrenic nerve, the emptying time was delayed more than six hours. In my cases all the stomachs with right phrenic paralysis in which fluoroscopic studies were made, emptied in from one to two hours. Following interruption of either left or right nerve, the small intestine lay in the pelvis.

In two cases symptoms and roentgenographic evidence of duodenal ulcer developed four and five months, respectively, following left-sided phrenic exeresis. In an experimental study of the development of peptic ulcer I pointed out the importance of the mechanical factor in the production of peptic ulcer. I believe it is entirely possible that the mechanical changes which took place in the stomach and duodenum following paralysis of the left hemidiaphragm were responsible for the subsequent development of the duodenal ulcers in these two cases.

One patient had a very severe gastric hemorrhage two months after a right phrenic interruption had been repeated. The only abnormality which could be found in this patient was that the stomach was changed in position as described above (Fig. 4). The hemorrhage in this case can be explained by the findings of Stanbury, in which at autopsy he found that the mucous surface of the dilated duodenum was congested and weeping blood.

Paralysis of the right hemidiaphragm causes the position of the gall bladder to be higher and more transverse than normal. In 4 of the patients the gall bladder did not completely empty after a fatty meal. In 3 of these cases, cholecystectomy was performed. In none

of the 3 patients was the gall bladder found to be definitely pathologic. Two of the patients, however, stated that they were much improved following the operation.

Of the 173 patients studied, there were 41 patients with temporary phrenic interruption and 14 patients with permanent phrenic exeresis

Fig. 1. Fig. 2.

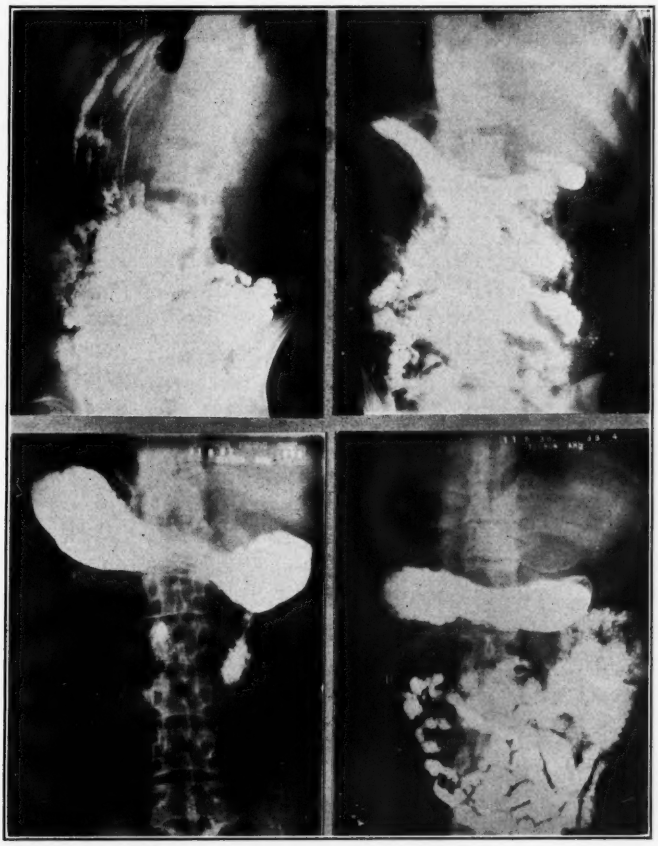

Fig. 3. Fig. 4.

Fig. 1.—Roentgenogram of stomach following left phrenic exeresis. Note the dilated fundus and the pylorus bent at an acute angle.

Fig. 2.—Roentgenogram of stomach following right phrenic exeresis. Note the transverse position of the stomach with the narrowed fundus.

Fig. 3.—Roentgenogram of stomach following a repeated temporary phrenic interruption. Note the pylorus shifted to the right and the hourglass appearance.

Fig. 4.—Roentgenogram of stomach following temporary phrenic interruption. This patient had a severe gastric hemorrhage. Note the transverse position of the stomach and the absence of a gas bubble in the fundus.

who had gastrointestinal symptoms. In none of the cases with temporary phrenic interruption did the gastrointestinal symptoms persist after the function of the diaphragm had returned. There were 4 cases

in which the symptoms recurred following a second phrenic interruption. While it was not possible to follow all the cases with permanent phrenic exeresis, 9 of the 14 patients still had symptoms when they left the sanatorium. In 2 cases in which a duodenal ulcer developed there had been permanent left phrenic exeresis. There was another patient who was known to have symptoms three and one-half years following his operation. In still another case, the patient brought a damage suit against the railroad two years after a right phrenic exeresis, on the grounds that he suffered continuously from stomach trouble as a result of the operation. It would seem that the danger of serious and persistent gastrointestinal disturbances following phrenic exeresis is another argument in favor of the use of temporary phrenic interruption.

SUMMARY

Following phrenic nerve interruption, gastrointestinal disturbances of clinical significance occur in about 25 per cent to 50 per cent of the cases. The syndrome following paralysis of the left hemidiaphragm is characterized by decreased appetite, feeling of fullness often associated with nausea and vomiting. The syndrome following paralysis of the right hemidiaphragm differs in that it is characterized by pain in the right upper quadrant. Roentgenograms taken before and after phrenic interruption show anatomic changes in the abdominal viscera of sufficient degree to account for the symptoms. Temporary phrenic interruption is advised in preference to permanent phrenic exeresis, because in all the observed cases the symptoms did not persist after the function of the diaphragm returned. If the possibility of gastrointestinal symptoms following phrenic nerve interruption is kept in mind, unnecessary concern over the patient's condition, and even useless operations, may be avoided.

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DISCUSSION

DR. JULIAN A. MOORE, Asheville, N. C.—I am glad that I have been fortunate enough not to have so many of these distressing symptoms occur after the operation of phrenicectomy, or at least they have not been called to my attention. Dr. Harper's work, however, certainly proves that it does occur and that definite changes are produced in the stomach, and this is another argument for temporary phrenic nerve interruption.

I have one incident that is rather curious and which I have never been able to explain that occurred in a patient in whom a right-sided phrenic neurectomy was done. Previous to the operation, she was constipated. After the operation, the constipation was markedly improved. I do not know how to explain that.

I have had a few patients who have complained of moderate gastric symptoms, mainly gaseous indigestion. In those patients in whom the operation was temporary, the symptoms disappeared when the function of the diaphragm returned, and in the others the symptoms have remained more or less permanent.

The incidence in Dr. Harper's experience is much higher than that of mine, but even then I think that the operation offers the patient many advantages in selected cases, but it certainly is an argument against indiscriminate phrenic neurectomies such as were done over a period of years.

DR. HARRY R. DECKER, Pittsburgh, Pa.—May I show one slide? There was a 42-year-old patient who had a phrenic nerve exeresis on the left side for cavitation and infiltration of the upper lobe.

In this particular case, a very high rise in the diaphragm was obtained, and infiltration disappeared to a large degree, but not completely. The patient developed intermittent dysphagia without any apparent indigestion. She did not notice it more than once a week, but it was an annoying symptom. It happened that as the tuberculosis progressed, it was necessary to do a thoracoplasty, and the thoracoplasty cured the dysphagia completely. Fortunately, the patient did not develop gastric dilatation.

DR. HARRY C. BALLON, Montreal, Canada.—In 1930, Wilson, Singer, Graham and I published some observations upon the esophagus, stomach, and heart following phrenicectomy. We also reported the occurrence of cardiospasm following phrenicectomy, but could not reproduce the condition in the dog.

Many of our findings are similar to those reported by Dr. Harper. I rather doubt, however, whether others are able to report as high a percentage of gastrointestinal disturbances following phrenicectomy. Many of these disturbances are of a transient character. I would agree with the statement that a temporary phrenicectomy is preferable to a permanent one on many occasions. I cannot accept the roentgenologic evidence presented as being typical or suggestive of so-called hourglass stomach. We observed deflections of the lower thoracic portion of the esophagus, increased

angulation of the abdominal portion of the esophagus, permanent large stomach bubbles, and contraclockwise rotations of the stomach. If after the contractile power of the left diaphragm is lost the individual continues to take large meals, air, which nearly everybody swallows during the act of deglutition, may become trapped in the stomach and cause discomfort.

In the experimental animal, as already stated, we were unable to demonstrate the occurrence of so-called cardiospasm. We did observe some widening of the esophagus.

Sauerbruch and others considered the sling of the diaphragm to be all important in the production of so-called cardiospasm. We were unable to corroborate this fact.

We also observed that some of gastric complaints following phrenicectomy were associated with complaints referable to the heart. I refer to the so-called gastrocardiac complex.

I would agree that gastrointestinal symptoms can occur following phrenicectomy, also that they do occur more frequently following permanent than after temporary phrenicectomy. The incidence of severe persistent gastrointestinal symptoms is not great. I feel that by far the greatest discomfort is occasioned by a collection of gas under the left diaphragm. We did not observe any ulcers or any of the serious lesions reported by Dr. Harper.

Although phrenicectomy is not the cure-all that many would still have us believe that it is, nevertheless it is a very useful procedure. Those who have not employed it widely should therefore be made cognizant of the fact that Dr. Harper's experiences are probably unique regarding frequency of persistent and distressing symptoms postoperatively.

DR. STUART W. HARRINGTON, Rochester, Minn.—I enjoyed Dr. Harper's paper very much. It is a subject I have been interested in for a great many years. About twelve years ago I first utilized phrenic nerve interruption in repairing diaphragmatic hernias. The procedure is utilized in those cases of diaphragmatic hernia where there is a large defect to be repaired; the relaxation of the diaphragmatic muscle following the phrenic nerve interruption permits the defect to be repaired and often obviates the more radical procedure of narrowing the diameter of the thorax by extrapleural rib resection.

I have found that a small percentage of patients who have had a permanent phrenic nerve interruption for repair of diaphragmatic hernia, have developed gastric symptoms. It was difficult to determine whether the symptoms were the result of the phrenic nerve interruption or whether they were related to the herniation of the stomach. In order to differentiate and to prove whether the symptoms were complications following the hernia or the result of the phrenic nerve interruption, I reviewed the cases of eventration of the diaphragm which have been seen at the Mayo Clinic and found that these patients have a gastric complaint which is rather indefinite in character and which I believe is undoubtedly due to the paralyzed elevated diaphragm which permits distortion of the stomach and lower end of the esophagus. Because of this observation, in the last seven or eight years I have utilized a permanent phrenic nerve interruption only in cases of diaphragmatic hernia where the defect was so great that I felt there would be a recurrence if the muscle were not permanently paralyzed. In most cases I have done a temporary phrenic nerve interruption which permits the diaphragm to return to normal activity between three and six months.

I believe that this is an important observation and that it should be recognized that there is a possibility of developing gastric complaints following a phrenic nerve interruption. In most instances the symptoms are rather mild and rarely develop to the degree of moderate severity; also, they are of minor importance when

compared to the beneficial effects which the procedure produces for the more serious conditions for which it is utilized. The percentage of patients presenting gastric symptoms following phrenic nerve interruption that Dr. Harper has reported is much greater than that which I have experienced.

DR. ALTON OCHSNER, New Orleans, La.—Dr. Harper referred to four cases of gastric dilatation following the thoracoplasty and associated with phrenic nerve operations. I should like to ask him whether those patients had symptoms following the phrenic nerve operations or after thoracoplasty. It has been our experience that thoracoplasty is frequently followed by gastric dilatation, which we felt was due to the stimulation of the splanchnics in the retropleural space.

As we all know, pneumonia is very frequently associated with gastric dilatation or ileus, and it has been thought by most people that it is a toxic manifestation. It is our opinion that it is due to a stimulation of the splanchnics secondary to pleural involvement. If measures are taken to combat this overstimulation of the splanchnics, the gastric dilatation can be easily taken care of.

DR. DUANE CARR, Memphis, Tenn.—I am impressed with this group of data which show that symptoms do occur, and give an explanation of their cause, following phrenic nerve interruption.

Up to the present, I have heard no mention of therapy in these cases. It is amazing to me that so large a percentage of the cases shown by Dr. Harper exhibit these symptoms. This has not been true in my practice. At least the symptoms have not been of sufficient intensity for the patient voluntarily to call them to my attention.

Without denying in the least that there is a definite physiologic basis for symptoms, I wonder if it is not true that suggestion plays a large part in the intensity of their manifestations. It is worthy of note that all these patients were in the same institution, and we are all well acquainted with the rapidity with which ideas circulate through a sanatorium. It is also worthy of note that two of Dr. Harper's patients were relieved of symptoms after the removal of normal gall bladders.

In my own practice, a patient appeared who had received a permanent phrenic nerve exeresis elsewhere and who was complaining bitterly of gas, severe gastric pain following meals, and recurrent gastrointestinal distress.

After a very careful and thorough examination, including gastrointestinal studies, no definite pathology could be demonstrated. I therefore referred this patient to a capable psychiatrist who, after going over him thoroughly, assured me that if this patient could be convinced that his diaphragm was moving again, his symptoms would probably disappear. So, unethical as it may be, I took steps to convince this patient that his diaphragm was functioning, even to the extent of holding a very poor mirror out in front of a fluoroscopic screen, asking him if he did not see the movement, which he did. I assured him he would continue, of course, to have a small amount of gas, and possibly a little indigestion, but that he would have no further severe pain, that it would rapidly disappear, which it did.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I would like to agree with Dr. Carr about this. I do not question the statistics that are presented in the paper, except that they are very unusual. We started doing a great many phrenic nerve operations thirteen years ago, and all were permanent exeresis operations. We assured the patients that it might help their lungs, might do them some good, but if it did not, it certainly would not do them any harm. As we look back, we see that our method was based on ignorance of future results, and a few patients have developed permanent gastrointestinal disturbances. In the first two or three years, we

did approximately a thousand phrenic nerve operations. This was before Dr. O'Brien had started to collect an abundance of statistics. Now, a thousand phrenicectomies is a mere drop in the bucket.

I am not substituting for Dr. O'Brien in making this comment, and I have no statistics. I don't know how many we have done now, because we have settled down, the same as everybody else in the country has, and we are doing temporary phrenicectomies almost entirely, and are not doing them indiscriminately as we used to. We try to choose cases. We perform the operation when we think it will be of help.

I am sure that out of that first thousand, or all the number we have done since, our percentage of complaints is very small, and nothing like Dr. Harper's report.

As to the reputation of the operation getting around that after the patient has the phrenic nerve cut he is going to have stomach trouble, it certainly does go from patient to patient; and if they are going to expect trouble, they may get it. Dr. Harper shows x-ray pictures which demonstrate that there is a change in the position of the stomach, but I know we had no such bad results here. There may be an occasional one, but nowhere near that. Our patients are not gathered together in institutions. Some are. There is a group at Trudeau and a group at Ray Brook, but the whole town of Saranac Lake is full of patients. They are in cottages. There never was any propaganda, of course, that "you are going to have belly-aches after your phrenics," and I do not believe they have had them. I just cannot believe that our percentage was anywhere near Dr. Harper's.

DR. RICHARD H. MEADE, JR., Philadelphia, Pa.—Last year I had an unfortunate experience with a patient who had been quite ill from tuberculosis and on whom a phrenic nerve operation was performed. This patient developed definite gastrointestinal symptoms which became progressively worse. The disturbance seemed to activate the disease in the lung. She went steadily downhill and died some months later.

I was so impressed with the effect of this left-sided phrenic that I determined in all future left-sided cases I would do a temporary operation. I started then to inject the nerve with novocaine, believing that if the symptoms produced by the operation were sufficient to cause later trouble, a paralysis lasting six months might be too long to wait for the return of function.

I did this temporary block on a number of patients. I was amazed to find that injection of one per cent novocaine was sometimes sufficient to allow paralysis lasting three days. These patients were observed very closely and fluoroscoped each day. In none of these patients did any gastric symptoms develop. Accordingly, this truly temporary procedure was converted into a slightly more permanent one several weeks later. I was amazed at the terrific pain experienced by the patients when the nerve was again exposed, so the interval was lengthened. I then wrote to Dr. Yates, whom I knew had been interested in temporary paralysis, and asked him about his experience. He had tried the same procedure with cocaine several years before, but had given it up because in his rather large series of cases he had found that symptoms due to any demonstrable changes following rise of the diaphragm occurred in too small a percentage of cases to worry him.

I want to ask if Dr. Harper has attempted to attack the problem from this standpoint of inducing truly temporary paralysis, one of a few days or of a few weeks.

DR. FRED R. HARPER, Denver, Colo.—First I want to thank you all for your very interesting discussion. I shall try to answer the various questions.

In regard to Dr. Moore's case in which he mentions that constipation was relieved after a phrenic nerve operation, we observed the same effect in one or two patients.

I do not have time to quote Stanbury's excellent article, but I should like to refer you to it. He goes further and shows the effect of phrenic nerve operations upon the bowel. He points out that after paralysis of the phrenic, the small intestine lies in the pelvis. You can see in Fig. 4 that the bowel is definitely lower in position. It may be that the altered position of the intestine caused the peristalsis to be increased and relieved constipation. I do not know that the explanation is correct; it is only a guess.

Dr. Ballon brought up the question of the hourglass deformity. I tried to be rather careful and not call the observed phenomenon an hourglass deformity, but said it appeared like an hourglass deformity. I do not think that there is a definite deformity. What happens is that half of the stomach moves while the other half does not; this change in position causes the stomach to be turned or rotated, giving the appearance of an hourglass deformity.

The question of the incidence of the symptoms being too high has been brought up by almost every discussant. Dr. Alexander called that to my attention when he first looked over my paper in Tucson and asked me to try to evaluate the symptoms so that I would not discourage the use of phrenic operations. For that reason I tried to show that the symptoms were not severe in most of these cases, but that they could be discovered by careful questioning. There were only 25 per cent, or less, in whom I thought the decrease of appetite from the left-sided phrenic operations or the pain from the right-sided phrenic operations were interfering with the progress of the patient. In many of the others the history was obtained after questioning the patient.

In regard to Dr. Carr's suggestion that the word travels through the grapevine route in the sanatorium, I think that is entirely possible, for we know that that does happen, especially with patients staying in a hospital for a year. However, the presence of gastrointestinal symptoms occurring after phrenic operations was first brought to my attention by the patients. When I would propose a phrenic nerve operation, the patient would suggest that he probably would not be able to eat as well, or that he would have stomach trouble if he had the operation.

There was one patient in our group who was an attorney for the Southern Pacific Railroad, a very intelligent man. He had been communicating with patients in other sanatoria in California and over the rest of the country. He told me it was common knowledge in other sanatoria that gastrointestinal symptoms occurred after phrenic operations. He more than anyone else called the phenomenon to my attention.

Although discussion of the possibilities would lead into the realm of theory, there are two points that I would like to make in regard to the possibility that the symptoms in the gall bladder cases were relieved after cholecystectomy by the power of suggestion. First, there was alteration in the function of the gall bladder because it did not empty after a fatty meal. I was completely fooled in all these cases in which I removed the gall bladder, since I operated not only for symptoms but also because of dysfunction of the gall bladder. Second, I believe it is conceivable that the change in position of the liver and the gall bladder may cause sufficient tension on the common duct or angulation of the cystic duct to produce these symptoms. When the gall bladder was removed, the tension on the common duct was relieved, and the patient thus relieved of his symptoms. That is purely theoretical, but I think it is possible. I do not know how you could prove it in the human being because x-ray pictures do not offer satisfactory proof and we rarely obtain autopsies soon after phrenic operations. I did not observe any marked changes in the biliary tract at the time of the operations on the gall bladder.

Dr. Ochsner asked me to explain the situation in the four cases that Dr. Thomas and I reported a year ago. In these four patients we did not have a history of gastro-

intestinal symptoms before the thoracoplasty. What happened in these cases was that after the thoracoplasty, the patients apparently did not do well. In one case it was rather dramatic. We thought we had entrapped air in the chest, so we tried to aspirate the air from the chest; instead, we aspirated gas from the stomach. The stomach had risen to the second intercostal space. As we mentioned in that paper, we lost two patients before we realized the situation. After that, we decompressed the stomach according to Dr. Wangensteen's method and the patients were relieved of their distress.

Joannides has mentioned that acute dilatation of the stomach may follow interruption of the phrenic nerve and, also, that it may follow operations on the lung or pleura. I think possibly the combination of the phrenic and thoracoplasty on the left side in these four cases brought about the acute dilatation of the stomach.

In regard to Dr. Meade's question as to whether we had tried temporary phrenic paralysis of very short duration, I would say we have not. As a matter of fact, I doubt if gastrointestinal symptoms would be liable to develop after a phrenic of very short duration. The majority of our patients with severe symptoms developed their symptoms rather late; generally, not immediately after the operation.

I was interested in Dr. Meade's suggestion because I think the method of doing temporary interruptions of very short duration should be a valuable asset not only in trying to determine if gastrointestinal symptoms would follow but also in determining other contraindications before doing a phrenic operation in certain cases.

AN APPRAISAL OF CLOSED INTERNAL PNEUMONOLYSIS IN PULMONARY TUBERCULOSIS

Relation of Tuberculous Empyema to Operation

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IN THE twenty-four years that have elapsed since Jacobaeus first described his method of severing intrapleural adhesions, hundreds of patients have been so treated. Moore[1] was able to collect 1,850 cases which he reported in 1934. Since that time probably there have been as many more cases. The operation has reached a well-recognized and well-deserved place in the treatment of pulmonary tuberculosis.

The most important complication which occurs following pneumonolysis is the development of pleural fluid, either serous or purulent. There has been a tendency to place the blame for this complication on the operation or the operator. This may be partly a just criticism, although the fact remains that most pneumothorax patients have fluid at one time or another and that a certain percentage develop tuberculous empyema spontaneously. The present paper is a discussion of this problem.

In discussing effusion, following artificial pneumothorax, Weisman[2] reported from collected cases that clear effusion occurred in from 17.1 per cent to 70 per cent and purulent effusion appeared in from 5 per cent to 21 per cent of cases. He found that fluid makes its appearance in 75 per cent of cases in the first six months and fluid appears in 91 per cent of all pneumothorax cases in the first year. The third month seems a particularly apt time for patients to develop effusion. This is an important observation because it is near this period, or soon after, that most pneumonolysis operations are performed.

Matson[3] believes that nearly all pneumothorax patients develop a pleural effusion at some time during their pneumothorax treatment, if it is continued long enough. He feels that the mere presence of gas in the pleural cavity, acting as a foreign body, and the unphysiologic intrapleural pressure conditions are sufficient to produce an effusion. In his series of 480 pneumothorax patients, 12 per cent developed tuberculous empyema. He does not explain how the serous fluid, which he attributes to pressure, can become purulent and contain tubercle bacilli. Leaver and Hardaway[4] report that 40 per cent of

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their cases of tuberculous empyema developed from clear fluid in the absence of any demonstrable spontaneous pneumothorax. Smart[5] concluded that artificial pneumothorax increased the incidence of pleural effusions, but the fluid in such cases was more amenable to treatment than that which followed other etiologic factors, e.g., spontaneous pneumothorax.

The cases on which the present discussion is based number 251 separate pneumonolyses on 230 patients. The operation has been performed by the high frequency current and the Davidson thoracoscope in every instance. The patients operated upon, both white and colored, were under treatment at seven different sanatoria.

The following indications have been recognized for the operation: (1) the presence of adhesions which prevent the closure of a cavity or the collapse of a heavily infiltrated zone after an adequate trial by pneumothorax alone; (2) symptoms, the result of pressure in the pneumothorax cavity, of sufficient severity to justify operation; (3) large, well-organized adhesions which contract and threaten the complete loss of the pneumothorax. Practically all authors have reiterated the opinion that the mere presence of intrapleural adhesions, as seen on the roentgen film, is not sufficient reason for cutting these adhesions. I wish to subscribe most heartily to that statement. If a clear indication for pneumothorax exists, it is particularly imperative that the pneumothorax should be an effective one. Every physician who deals with pulmonary tuberculosis realizes that occasionally in apparently almost hopeless cases brilliant results are obtained by a satisfactory and effective pneumothorax. The division of the compromising adhesions under local anesthesia can add little additional risk in such a situation.

TABLE I

CLOSED INTERNAL PNEUMONOLYSIS

Total operations	251
Number of patients	230
Explorations without operation	19
Cavities closed by operation	74.8%
Sputum changed from positive to negative by operation	93.5%
Satisfactory collapse	86.4%

Most of the patients reported had been receiving pneumothorax refills for from four to six months or longer. Operation on patients whose pneumothorax is of shorter duration than four months is occasionally justified if the adhesions are of such nature that closure of the cavity seems impossible with prolonged pneumothorax alone.

Apparently the only valid contraindication is the existence of an acute pleurisy with fluid, either serous or purulent. Successful operations can be done in chronic afebrile cases of pleurisy with effusion, provided the adhesions are not covered with fibrin and can be clearly

seen. It is assumed that the adhesions are of the type which can be cut, without danger of injury to the lung, and without excessive risk of hemorrhage.

The general results, exclusive of complications, have been most satisfactory (Table I). There have been no deaths attributable to the operation. Of those patients having cavities, 74.8 per cent of the cavities were closed. A satisfactory collapse was obtained in 86.4 per cent of patients operated upon, which is almost identical to the percentage of patients with satisfactory collapse as reported by Welles.[6]

Besides the development of serous fluid or pure tuberculous empyema, the complications that developed in association with operation are the following:

TABLE II

COMPLICATIONS

Hemorrhage, severe	1
Hemorrhage, moderate	2
Extensive subcutaneous emphysema (loss of lung collapse)	1
Pulmonary (bronchial) hemorrhage	2
Pyogenic or mixed empyema	0
Tuberculous empyema	6 (2.6%)
Demonstrable fluid before operation	64 (27.6%)
Demonstrable fluid after operation	78 (33.6%)
Mortality	0

Two of the cases of intrapleural hemorrhage required open operation for control. Except in the case of the patient who lost the pneumothorax, none of these complications seemed to have disturbed the favorable course. Pyogenic empyema did not occur in any case. No patient presented an immediate postoperative flare-up in the contralateral lung.

The incidence of pleural fluid, serous or purulent, after operation is strikingly low. Sixty-four patients (27 per cent) had fluid in the pleura at the time of operation. Fourteen additional patients (6 per cent) developed fluid after operation, mostly within a few days. Among the total of 78 patients (33.6 per cent) who had fluid at one time or another, in 6 patients the fluid became purulent; 4 of the 6 had fluid at the time of operation. Parenthetically, it may be noted that in 270 individual thoracoscopic examinations there was encountered only one case presenting multiple pleural tubercles, similar to the multiple tubercles so commonly encountered in tuberculous peritonitis.

It is obvious that the total incidence of clear fluid and of tuberculous empyema in the present series is well within the limits of corresponding incidence to be expected from pneumothorax alone. This report, therefore, presents evidence that pneumonolysis is not an important factor in the development of intrapleural fluid.

An analysis of the cases of tuberculous empyema appears in Table III. The first 4 cases are possibly open to the criticism that the patients were operated upon too soon after the institution of pneumothorax. Each of the 6 patients had a large cavity and the sputum was heavily loaded with tubercle bacilli. One was a child of 14 years. Closure of the cavity was imperative in each case. Four of the patients had one operation each and 2 patients had two attempts to divide the adhesions. In spite of the development of tuberculous empyema, the cavity was closed in 5 of the 6 patients. All these patients are doing well at present. Only one patient developed tuberculous empyema within less than five months after operation.

TABLE III

TUBERCULOUS EMPYEMA

CASE	DURATION OF PNEUMOTHORAX BEFORE OPERATION	FLUID BEFORE OPERATION	TIME AFTER OPERATION EMPYEMA OCCURRED	CAVITY CLOSED
I	3 mo.	none	6 mo.	Yes
II	3 mo. 5 mo.	small amount	9 mo.	Yes
III	2 mo. 4 mo.	none small amount	9 mo.	Yes
IV	2½ mo.	none	3 mo.	No
V	5 mo.	small amount	30 mo.	Yes
VI	8 mo.	small amount	5 mo.	Yes

All patients had positive sputum and large cavity. Each patient had purulent fluid with tubercle bacilli. Case IV abandoned pneumothorax—had phrenic paralysis later.

A comparison of the incidence of tuberculous empyema with the source sanatoria presented interesting results. Five of the 6 cases of tuberculous empyema came from one institution which has a high proportion of patients with moderate and far advanced pulmonary tuberculosis. Among 99 patients operated upon from this institution, 5 developed empyema; whereas from an institution caring largely for patients with minimal and moderately advanced lesions, no empyema developed in 90 patients operated upon.

For completeness, a final postoperative event must be mentioned, namely, the rupture of a cavity into the pleura. Three of these occurred, followed immediately by death. As they occurred at intervals of three and one-half months, four and one-half months, and nine months respectively, they have not been ascribed to operation. This seems justifiable because such rupture has frequently occurred with pneumothorax alone. As a postoperative event, Dufault and Laroche[7] reported 6 autopsies in 9 cases. In all the autopsies the point of rupture was at a distance from any of the divided adhesions.

DISCUSSION

Although, as has been said, the evidence here presented does not suggest that pneumonolysis is the cause of any important number of effusions into the pleura beyond those that would occur following the institution of pneumothorax alone, it may be profitable to discuss the possible mechanisms by which the operation might cause effusion. Most authorities agree that the fluid, serous or purulent, that is seen in the presence of pneumothorax contains tubercle bacilli. They believe that in most instances it is a true exudate and not a transudate. The possible sources of these bacilli in pneumothorax, without or with division of adhesions, has been discussed by several writers.

Jaffe[8] states positively that tuberculosis of the pleura is always secondary. It reaches the pleura by direct extension from a focus in the lung. In some instances it can be traced to a caseous tuberculosis of the chest wall or of the thoracic lymph nodes. It has been shown by William Snow Miller[9] that tubercle bacilli can be carried to the pleura by way of the lymph and blood vessels.

Lemon[10] has shown experimentally that some tubercles are ineffectively bounded by the visceral pleura, and tubercle bacilli, both free and intracellular, were present in large numbers and had free access to the pleural sac. Cellular masses attached to the pleural surfaces contained large numbers of tubercle bacilli. Tubercle bacilli were also found in loose lymph spaces. He reported definite evidence of invasion of the pleura, subpleural space, and pulmonary parenchyma following the intrapleural injection of tubercle bacilli in rabbits. Fluid appeared in increasing amounts nine days after the injection of bacilli, and massive effusions appeared within four weeks. Massive purulent effusions occurred only in sensitized animals which had received a large sensitizing dose of virulent bacilli. The effusions became massive because of two factors: the pleura was constantly being subjected to reinfection by the autogenous addition of relatively small numbers of bacilli, and fluid was not being absorbed as readily as it normally would be because it was continually re-forming and because the lymphatic system was blocked by tuberculous tissue throughout.

These findings are extremely important because they show conclusively that tubercle bacilli can readily gain access to the pleural space. The lymphatics contain bacilli, and it is inconceivable that all lymphatics are sealed over when the adhesions are divided. In addition to this, exudative plaques on the visceral pleura often contain tubercle bacilli. Such plaques and clumps of fibrin near adhesions are very likely to be disturbed and displaced mechanically by the instrument, even though no cutting is done at that point, and tubercle bacilli thereby freed in the pleural space.

By special methods of study, Joannides[11] has been able to demonstrate tubercles on the surface of the lung, which are ordinarily unnoticed because of their small size. Such tubercles near the edge of the lung are capable of dislodgment into the free pleural space. When pneumothorax is started, and the lung is peeled away from the chest wall, many tubercles may be freed. Joannides also suggests that the respiratory variation in fluid level in effusion cases and the mechanical washing of the pleural surfaces may dislodge additional tubercles.

Cross section of an area of fused thickened pleurae may show a narrow grayish zone between the pleurae, which is a chain of more or less confluent tubercles. If such an area is tremendously stretched, as may occur in the formation of adhesions with the institution of pneumothorax, it may be cut across in dividing adhesions with the consequent liberation of tubercle bacilli into the free pleural space. Thus tuberculous empyema can occur without injury to the lung.

Judging from the foregoing rather conclusive evidence of the presence of tubercle bacilli in probably all cases of pleural effusion, it would appear that tuberculous empyema is a condition which may occur following pneumonolysis, even in cases in which it is clear that pulmonary tissue has not been injured.

Fluid, therefore, which forms after the severance of intrapleural adhesions is probably mainly due to three factors: the response of the pleura to injury; perhaps a sensitizing reaction due to the release of additional tubercle bacilli; and actual infection and invasion of the pleurae by tubercle bacilli which were freed at the time of operation.

From this presentation of the possible mechanisms for the introduction of tubercle bacilli into the pleura by operation, it is not to be concluded that the procedure offers a real danger of this event. As already stated, the present figures suggest that the danger is very slight, in fact, that the operation does not add materially to the incidence of effusion occurring commonly with pneumothorax alone.

It is evident from the experience of those caring for large numbers of pneumothorax patients that tuberculous empyema which occurs in the absence of spontaneous pneumothorax, or without injury to the lung during the severance of adhesions, is not a serious complication if promptly and properly treated.

CONCLUSIONS

The incidence of serous fluid (6 per cent) and tuberculous empyema (2.6 per cent) in this series of 251 pneumonolyses is considerably less than that occurring in ordinary pneumothorax.

It is evident that the operation may contribute to the development of tuberculous empyema by freeing tubercle bacilli into the pneumo-

thorax space, where they were most probably present prior to operation. This does not need to be a deterrent in patients who are in real need of an effective pneumothorax.

Fluid after pneumonolysis occurs in not more than the percentage of cases in which it is expected following pneumothorax alone. Its occurrence, therefore, cannot be attributed wholly to operation.

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DISCUSSION

DR. JULIAN A. MOORE, Asheville, N. C.—I enjoyed listening to Dr. Drash's comprehensive discussion about the formation of fluid. I do not think, however, that he is quite fair to the operation to name a tuberculous empyema that occurred three to six months after the operation as a complication; it does not seem to me, unless the fluid occurred immediately, that the occurrence of an empyema from six to thirty months later could be ascribed to the operation.

We certainly know that tuberculous empyema occurs in pneumothorax without any operation, and at any time, very unexpectedly. The spontaneous rupture of the lung that occurs more than six weeks after the operation could certainly not be ascribed to the operation, and should not be considered as a deterrent.

In regard to the contraindications to the operation, I am convinced, from three sad experiences that I have had, that when a patient has bilateral disease, and is hopelessly ill, a sympathetica and ill-advised attempt to make a satisfactory collapse on one side should not be attempted. The only three cases that ever caused me trouble were of this type.

I, too, have had one patient lose his pneumothorax by a subcutaneous empyema which I could not stop.

DR. HANS C. JACOBAEUS, Stockholm, Sweden.—It has interested me very much to hear this paper on pneumonolysis, and I am very glad to hear of the research that has been done. I have had some difficulty in following the paper; therefore I will only tell you a little of my own experience.

Where you have surface adhesions, you must loosen a large part of the lung. I often do an operation early, one or two months after inducing pneumothorax. When one is operating on old cases, one finds much more fibrous tissue.

As to hemorrhage, I have in my material six to seven hundred cases; only twice had severe hemorrhage occurred, and that was observed immediately after the operation. First the patient was very pale, and when the pleura was examined it was found to be filled with blood.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I have one or two points I should like to add. In the first place, I should like to agree with Dr. Jacobaeus about the value of early operation. I have had numerous cases presented to me for

pneumonolysis in which a series of x-ray films were shown and a rather difficult operation was presented. In the series we could go back several months to a time much closer to the institution of the pneumonolysis when we could have done a very easy operation.

The common progress of adhesions is not for them to stretch, thin out, and get easier to cut. That can occur, and occasionally with high pressure pneumothorax, I am sure some adhesions do actually rupture, but most people carry out pneumothorax without high pressures, and the great tendency is for the lesions to thicken and shorten and toughen as time goes on.

I think it is far better to go ahead and do the pneumonolysis promptly, if you think it is going to be indicated, and give the patient the benefit of that extra time with more full collapse of the lung, or complete collapse, than to put it off three or six or nine months without reason. I have done numerous cases early, within a month, and some even less than a month, after the institution of pneumonolysis, cases in which the lung has gone down very satisfactorily, except that the cavity was held out by one or two small adhesions.

I have never seen a bad result, or anything to indicate that that was not a good thing to do, or found any reason to wait a set period of time. I do not see the argument against it. The one argument that has been presented to me is that if it is done that early, the mediastinum may still be too flexible, where it will stiffen later, and that with the introduction of the instrument and the free flow of air into the chest through the instrument or the cautery cannula, there will be a rapid expansion of the lung. I have never seen that occur. It is possible, but I think in principle early operation is preferable.

The hemorrhages that Dr. Jacobaeus mentioned as not being discovered until the next day, could very well have come from the wound in the chest wall. I had one case in which that happened, and in which a slight trickle of blood from the chest wall at the entrance of the cautery cannula was observed at the time of the operation. It was so slight that it was deemed unnecessary to do anything about it, but I believe that after the cannula was removed and its pressure taken away, the intercostal vein continued to bleed, and it filled the chest cavity. The result was not fatal, and the patient made a complete recovery. I do not think it had anything to do with the burning of the adhesion. I have had one or two hemorrhages from adhesions at the time of operation. I do not count occasional dripping as hemorrhage. If the adhesion begins to drop slowly, it can be stopped in a minute by application of heat. I do not count that as a hemorrhage at all. I have had one or two cases which I feared I would not be able to stop. I have always been able to stop the hemorrhage, however, and have not had to resort to open operation, as Dr. Drash says he has twice.

As to the question of bleeding from the chest wall from the introduction of the instrument, I think that is unavoidable. It is well to look at the cautery cannula, if you are using a two-instrument outfit, to see if there is bleeding. You cannot, however, look at the site of entrance of your thoracoscope instrument unless you put one in on the other side, too. I think it is just as apt to occur in one place as the other. It is an unavoidable accident, I believe, that occasionally in rare instances you may injure the intercostal vessel, either the artery or the vein, probably the vein, and not stop that bleeding by the pressure of the cannula itself while you are working. That occurrence is so rare, however, that in the several hundred cases which I have had, it occurred only once, and that without a fatal result. I think it must be overlooked as a contraindication or a danger of the operation.

I quite agree with Dr. Moore about the entire lack of relation of pneumonolysis to tuberculous empyemas occurring three months to two years afterward, and also the relationship between spontaneous rupture of the lung and the operation. As he said, when the autopsies were done on these patients, the ruptures were found to

occur at a portion of the lung distant from or at least not at the adhesion, and I think that is true. Lungs do rupture, empyemas do occur, but when they occur at that distance, I think Dr. Drash is assuming too much. He is taking blame for something and trying to offer excuses for it when it is too far in the background.

DR. EDWARD J. O'BRIEN, Detroit, Mich.—A number of years ago, I read a paper, before this Society, on open pneumonolysis, in which I very strongly advocated the use of that procedure in preference to the closed pneumonolysis. I want to tell you I have changed my mind. The reason I favored it at that time was because of the fear of hemorrhage. In recent years, our instruments have been improved materially. We now use electrocoagulation and my fears of hemorrhage have been lessened to such an extent that we are routinely using the closed pneumonolysis. We are using the Davidson-Cutler scope, with a few modifications which I have made. The instrument has an examining scope which is used first and gives an excellent field; but when you put in the carrier with the cautery point, the view is not so good. I wrote and asked if we could not have the carrier put on the examining scope. They said it was possible but thought it would be too large. A sample was made and sent to me. It is not too large, and it provides a clear field. It is almost like an open operation through this scope, for of course there is direct vision.

I too think Dr. Drash is borrowing trouble when he talks about empyemas occurring late after the operation. In our series we do not feel that the incidence of tuberculous empyema following pneumonolysis is any greater than that we find in the regular run of pneumothorax patients in the institution.

DR. JOSEPH GORDON, Seaview, New York, N. Y.—In a recent review of approximately 200 cases at Dr. Coryllos' clinic at Seaview, I am very happy to say that our results almost parallel Dr. Drash's, so much so that we concluded that the complications were the complications of the disease and not of the operation. They differed in two instances. First, we had no hemorrhage, because in Dr. Coryllos' thoracoscope he uses the active cautery rather than the diathermy. The other difference is that we did have seven cases of mixed infection with resulting empyema; we concluded that these were probably the results of the operative procedure only because we were dealing with extensive bilateral cases where on one side no pneumothorax was possible. Also, we had attempted by extensive dissection to try to gain control at once of the contralateral lung and prepare the patient for a thoracoplasty on the opposite side.

In most instances we find that the fluid disappears in the first week.

DR. E. C. DRASH.—I wish to make it clear that all cases of fluid and tuberculous empyema were included for the sake of completeness. Tuberculous empyema, which developed thirty months after operation, cannot be attributed to that operative procedure. I do feel, however, that pneumonolysis probably has some bearing in some cases on the development of tuberculous empyema. The occurrence of tuberculous empyema in this series is not greater than that which would be expected from pneumothorax alone.

The comment has been made that originally the criteria for the selection of cases for pneumothorax were almost identical to the criteria which we now accept for thoracoplasty. In recent years pneumothorax has been instituted in cases with bilateral pulmonary disease and even with extensive tuberculous involvement elsewhere in the body. When patients with extensive pulmonary tuberculosis are given pneumothorax, the incidence of pleural fluid, both serous and tuberculous, is certain to increase.

I am sorry that no one discussed the observation concerning the infrequency of disseminated tubercles on the visceral and parietal pleura. Disseminated tubercles are rather a common finding in tuberculous peritonitis, but this condition appeared in only one of 270 thoracoscopic examinations.

TUBERCULOUS EMPYEMA

Report on 154 Cases

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THIS study comprising 154 patients does not represent my own series but is based upon consecutive cases occurring in the practice of local physicians and sanatoria. One hundred and thirty-four had their inception during the five-year period from 1929 through 1933. They were tabulated this spring so we have a follow-up of three to eight years. Twenty others are included, 6 of whom antedated this period, and only 3 began during 1936 and so have been followed less than a year.

Table I

PHYSICIANS	TOTAL CASES	FIVE-YEAR GROUP
Dr. H. M. Kinghorn	11	7
Trudeau Sanatorium	31	28
Dr. Henry Leetch	13	13
New York State Hospital (Ray Brook)	9	9
Dr. E. N. Packard	5	5
Jefferson County Sanatorium	4	3
Dr. Lawrason Brown and Dr. J. N. Hayes	36	32
Dr. J. Woods Price	13	13
Stony Wold Sanatorium	21	14
N. V. A. Lodge	2	2
Dr. Malcolm F. Lent	5	4
Dr. S. F. Blanchet	4	4
	154	134

For the purpose of this study, tuberculous empyema was defined as any turbid pleural fluid in the presence of pulmonary tuberculosis or any fluid containing tubercle bacilli on direct smear.

In the series, there were 47 known persistent bronchopleural fistulas or an incidence of 30 per cent. There were 42 known secondary infections. About half were infected from the onset by either rupture of the lung or contamination of the pleura. The rest began as straight tuberculous fluids and became infected later—in one instance after fluid had been present for eight years. It is interesting that of those patients who originally had a noncontaminated tuberculous empyema and who did not die of it (in other words of those who lived long enough to develop other complications) 30 per cent finally developed them

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either by perforation or by secondary infection. Others may later do so. Originally we had attempted to subdivide all empyemas according to the amount of underlying disease. In a compilation of this kind where the cases came from twelve separate sources, this subdivision did not prove feasible.

TYPE AND DEVELOPMENT

Table II illustrates the method of development. In 17 cases the empyema was not a complication of artificial pneumothorax. In 12 it began as pleurisy with effusion, and in 5 pneumothorax and empyema were ushered in by a spontaneous pneumothorax. During this five year period, only 8 cases followed pneumonolysis within a month and could reasonably be attributed to it.

TABLE II

DEVELOPMENT OF 154 CASES

PLEURISY WITH EFFUSION	SPONTANEOUS PNEUMOTHORAX	ARTIFICIAL PNEUMOTHORAX	FOLLOWING PNEUMONOLYSIS	POSTTHORACOPLASTY INFECTION	OTHER	UNCERTAIN
12	5	121	8	4	2	2

Postthoracoplasty infections accounted for 4 cases. In each instance thoracoplasty was being performed for the closure of a cavity over a pneumothorax without fluid. A wound infection developed which worked its way through the pleura and an empyema promptly ensued. In 3 instances, bronchopleural fistulas developed. In only 2 of the 4 cases was it ever possible to attempt to complete the thoracoplasty and it was successful in one.

The time of development of empyema is of interest and is shown in Table III.

TABLE III

TIME IN MONTHS OF DEVELOPMENT OF EMPYEMA AFTER ONSET OF FLUID

MONTHS	NO.	%
0- 6	93	67.0
7-12	26	19.0
13-36	13	9.5
37+	6	4+
	Uncertain—16	

This is taken from the time of the appearance of fluid and not from the date of initial pneumothorax. It will be seen that although 86 per cent became purulent within the first year, 6 of 138 cases appeared after three years.

Table IV gives us the number having a secondary infection. You will note that 12 patients with bronchopleural fistulas are listed as not having

any secondary infection. For this, there are several reasons: first, several died promptly after the perforations and before they had time to develop secondary infections; second, some had bacteriologic studies immediately after the perforation which were negative for secondary infections, and then none later. Those are recorded as not having mixed infection. In our series, most of the empyemas developed soon after the onset of fluid, but this was not true with the secondary infections.

TABLE IV

Type		
Tuberculous	*Mixed*	*Uncertain*
105	42	7
Bronchopleural Fistula		
Tuberculous	*Mixed*	*Uncertain*
12	31	4

(Bronchopleural Fistula, Total 47, Dead 30—63.8%)

TABLE V

TIME OF DEVELOPMENT OF SECONDARY CONTAMINATION IN THOSE SIXTEEN CASES KNOWN TO HAVE BEEN STRAIGHT TUBERCULOUS AT ONE TIME

YEARS	NO.	%
First year		
First half	4	25
Second half	4	25
Second	1	
Third	2	
Fourth	2	
Fifth	0	50
Sixth	0	
Seventh	2	
Eighth	1	

Table V is the tabulation of those 16 patients who are known to have had an uncontaminated empyema at one time and who later developed a mixed infection. Exactly half of them developed within the first year. Three cases developed seven to eight years after the onset of fluid. In other words, as long as there is fluid or pus in the pleural cavity, the hazard of secondary infection is present.

TREATMENT

I shall now present statistically the results of the various methods of treatment. For the purpose of tabulation, we have called empyemas without secondary infection Group A, and those with secondary infection Group B. There were only 6 patients who had no treatment and they were divided—2 dead, 2 resting, and 2 well and working.

The simplest method of treatment is aspiration with air replacement. Practically all our patients had at least diagnostic aspiration. There were, however, 46 who were treated by aspiration and their fate is shown by Table VI.

TABLE VI

NO TREATMENT

NO.	DEAD	RESTING	WELL AND WORKING
6	2	2*	2

ASPIRATION ONLY

	NO.	DEAD	RESTING	WELL AND WORKING NO.	%
Group A					
Tuberculous	38	15	8	15	39.5
Group B					
Mixed infection	6	5	1	0	
Uncertain	2	1	0	1	
Total	46	21	9	16	35.0

Of the sixteen well and working, the lungs of nine are presumably reexpanded.

*Includes one who was well and working with reexpanded lung until relapse in 1936.

In Group A, 40 per cent are living and working, with an equal number dead. Of the 15 supposedly well and working, apparently 9 have reexpanded lungs with obliteration of the pleural space while only 6 are carrying on with their empyema pockets, either with or without fluid. In Group B, represented by 6 individuals, there is only one living and he is not well or working.

TREATMENT WITH SALINE IRRIGATIONS OR THE VARIOUS DYES

The contrast in the two groups is again quite striking. Judged by the dead, and by the well and working groups, the patient with a mixed infection would seem to have about half as much chance as he enjoyed before the pleura was secondarily contaminated.

As to the treatment itself, of those classified as well and working, only 7 of the original 76 did not have further treatment. Of these, 5 were in Group A and 2 were in Group B. These dyes included all the common ones. On several patients, the physicians used the organic mercurial dyes synthesized in the Trudeau Sanatorium Research Laboratory under the supervision of Dr. S. A. Petroff.

Table VIII records our experience with oleothorax. In Group A of 22 patients exactly one-half are reported as well and working without further treatment. As a means of returning patients to work, this shares first place in our series with complete reexpansion of the lung, each method having 50 per cent in the well and working group, while

TABLE VII

TREATMENT WITH DYES AND/OR SALT

	TUBERCULOUS	MIXED	TOTAL
Number of patients:	43	33	76
Dead	15—35.0%	19—58.0%	
Well and working	14—32.5%	5—15.0%	
Resting	14—32.5%	9—27.0%	
	43	33	
Reported well and working:			
No further treatment	4	1	
Lung reexpanded	1	1	
Oleothorax	6	1	
Tube and/or thoracotomy	1	0	
Thoracoplasty	2	1	
Oleothorax and thoracoplasty	0	1	
	14	5	
Reported resting:			
No further treatment	4	1	
Lung reexpanded	4	0	
Oleothorax	3	1	
Tube and/or thoracotomy	2	2	
Tube and/or thoracotomy and thoracoplasty	0	2	
Thoracoplasty	1	0	
Oleothorax and tube and/or thoracotomy	0	1	
Oleothorax and thoracoplasty	0	2	
	14	9	

TABLE VIII

TREATMENT WITH OLEOTHORAX

	TUBERCULOUS	MIXED	TOTAL
Number of patients:	22	13	35
Dead	6—27.2%	6—46.2%	
Well and working	11—50.0%	3—23.0%	
Resting	5—22.8%	4—30.8%	
	22	13	
Reported well and working:			
No further treatment	11	1 (Oleothorax discontinued)	
Thoracoplasty	0	2	
	11	3	
Reported resting:			
No further treatment	4	1	
Tube and/or thoracotomy	1	1	
Tube and/or thoracotomy and thoracoplasty	0	2	
	5	4	

thoracoplasty in the same group is 44 per cent. The mortality rate, however, is three times as high as in complete reexpansion and half again as high as with thoracoplasty.

In Group B, it was of no value. Of the 13 patients in that group, we have listed 3 as well and working, but that is misleading. Two subsequently had thoracoplasty and the remaining patient, No. 51 in our series, had to be included in this group because he did have oleothorax, despite the note of the physician that oleothorax was tried and abandoned because it did not control the empyema. He reverted to washings and is now working with a chest partly full of fluid, presumably pus, after having had two flare-ups of mixed infections in the last several years. In our entire series this man stands alone.

Oleothorax then was very effective in Group A and of no value in Group B. Again it should be noted that as long as there is fluid in the chest, be it pus or oil, there is possibility that sooner or later it will rupture either into the bronchus or through the chest wall. This has happened several times in our series, and there is at least one young woman who, after the lesion ruptured to the outside and the rupture successfully healed, went on to reexpansion of the lung and is now in the working group.

Because of the ever present danger of perforation it would seem wise in the successful instances, once the infection is controlled, to attempt aspiration and carry on with a dry pleura.

TUBE AND THORACOTOMY

Table IX gives our experience with tube and thoracotomy. Many men attribute a majority of the mixed infections to injudicious drainage of a straight tuberculous empyema. We had 10 patients in Group A who had tube or thoracotomy, but 9 had a bronchopleural fistula and one a chest wall fistula, both of which we feel are indication for tube

TABLE IX

TUBE AND/OR THORACOTOMY

	NO.	WELL	LIVING	DEAD
Group A: 9 bronchopleural fistulas 1 chest wall fistula	10	1	2 (1 thoracoplasty)	7
Group B:	22	3 (3 thoracoplasty)	6 (2 thoracoplasty improved) (4 sick)	13 (2 thoracoplasty)
Uncertain bronchopleural fistula	1			1
	33	4 (3 thoracoplasty)	8 (3 thoracoplasty) (5 sick)	21 (2 thoracoplasty)

drainage if the fluid prevents the patient from sleeping or resting because of the constant cough and discharge. It will be seen that the death rate in this group is appalling. Twenty-one out of the 33 are dead and only 4 are well. Of these, 3 have had thoracoplasty. In the living and still ill group there are 8, of whom 3 have had thoracoplasty and are doing fairly well. The remaining 5 are listed as sick. Only one of the 5 sick can be listed as even in fair condition, and he is losing ground slowly. Of the dead, 2 had thoracoplasty.

THORACOPLASTY

Considering the proved effectiveness of thoracoplasty in the treatment of tuberculous empyema, I was surprised that we have only 28 instances of its use in this series, 16 and 12 in Group A and B respectively. There were two other instances of patients in this group having thoracoplasty, but they were in the original four cases where thoracoplasty over a pneumothorax ushered in the empyema, and the patients died promptly.

The other 2 weathered the infections following drainage and subsequently came into this group, one being cured, the other dying before complete collapse could be obtained.

In Group A we see that of 16 patients, 3 are dead, another will probably die soon because of an extensive spread to the good lung; but, on the other hand, we have seven patients well and working and of the 6 living, 3 were operated upon within the last year and are doing very well. It is reasonable to expect that several more will join the well group.

Group B presents the most difficult problem in any form of treatment. We have here 12 patients, with one-third dead and 5 well and working. Of the 3 living, one has had the last stage of a Schede operation within the last few months and seems destined within a year to take his place with the well and working. One other of these 3 I think might with justification go into the working group. His physician has reported him as "well, not working, but able to fish."

TABLE X

THORACOPLASTY

	WELL	LIVING	DEAD	TOTAL
Group A:	7—43.7%	6 (Three operated upon within last year	3—18.8%	16
Group B:	5—42.0%	3 (One operated upon within last three mo.)	4—33.0%	12
Total	12—43.0%	9	7—25.0%	28
Bronchopleural fistula, all included in Group A or B	6	4	3	13

Adding the two groups together, we find that 12 of the 28, or 43 per cent, are well or working, with an excellent prospect that there will be several additions within the next year. There are 7 dead, or 25 per cent. Undoubtedly a few more will drop into this group within the next few years, but at present it does not seem that there will be a great many.

At the bottom of Table X you will see a separate classification according to the presence of bronchopleural fistula, but you will notice that there is no significant change from the totals.

Among these 28 patients, two have had Schede operations performed after the completion of the thoracoplasties.

Of the 28, 20 were done locally; the other 8 were distributed, 2 by Dr. Archibald, one by Dr. Graham, one by Dr. Lambert, one by Dr. Coryllos, 2 others in New York (but the names of the surgeons are unknown to us), and one by Dr. Churchill.

FORCIBLE REEXPANSION

One reason for the inclusion of 14 cases following the termination of our five-year period is because of Stony Wold Sanatorium. Their treatment of this condition has changed completely since 1933. They now attempt forcible reexpansion of all of empyemas. Seven of the 14 cases are in this group. I think this series is too small from which to draw any sweeping conclusions. In addition, 2 cases from Trudeau are included, making a total of 9. There are 2 patients dead, 3 have had thoracoplasty, 3 are living with reexpanded lungs, and one is living partially reexpanded. There is this to be said about this method of treatment: once the pleural space is obliterated, empyema ceases to be a factor and the risk then becomes that of the underlying tuberculosis. It would seem that there are certain hazards, first, that in forcible reexpansion there is danger of the rupture of the tuberculous lung, and the mortality rate of ruptures in our series is 63 per cent. Second, even an incomplete pneumothorax will frequently compress portions of a diseased lung, with amelioration of symptoms. We have all seen certain patients become worse when we have attempted to abandon an incomplete pneumothorax, with the consequent reexpansion of diseased lung. In such an instance I believe primary thoracoplasty is preferable. The third danger is that prolonged high negative pressure may cause such marked thickening of the pleura that even a complete thoracoplasty will be ineffective. One such case is the one which was referred to Dr. Graham.

We have reviewed the various methods of treatment. Let us now look at the results. In the discussion of the death rate from tuberculous empyema, the question repeatedly arises whether the deaths are due to the empyema itself or to the underlying tuberculosis. In this series, we cannot definitely answer that question. We do know, how-

ever, that there are 76 patients in this series who throughout their entire illness never had more than a minimal lesion on the opposite side. The mortality in this group is 26 per cent. In other words, one out of every 4 patients in whom the infection never spreads to the opposite side and who presumably has at least a partial collapse of the pulmonary tuberculosis on the empyema side still dies of the empyema. This does not take into consideration those patients who at the beginning of the empyema have a minimal lesion or less on the good side and who, from the ravages of the disease, develop an extension of the tuberculosis on the good side and so automatically come out of this group.

TABLE XI

ENTIRE GROUP

STRAIGHT TUBERCULOSIS	DEAD NO.	DEAD %	LIVING RESTING NO.	LIVING RESTING %	LIVING WELL AND WORKING NO.	LIVING WELL AND WORKING %
Group A:						
105 cases	35	33.3	28	27.7	42	39.0
12 cases bronchopleural fistula	7	—	3	—	2	—
93 cases of tuberculosis without bronchopleural fistula	28	30.1	25	27.9	40	42.0
Of these 28 deaths two were not attributable to tuberculosis	2	—				
	26	28.0				
Group B:						
Mixed infection 42 cases	25	59.5	10	23.8	7	16.6

Table XI gives a résumé of our series as a whole. We have figured separately the percentages in the five-year group, but the discrepancies are so slight that we give only the one table.

By the various methods of treatment used we have in:

Group A (not including bronchopleural fistulas):

28% Dead of tuberculosis
42% Well and working

Group B:

60% Dead
17% Well and working

Of those that are well and working, exactly one-half have had thoracoplasty or have completely obliterated the pleural space; another 8 are continuing with oleothorax; only one-third of the well group are continuing with pneumothorax or with fluid. At this point, it should be noted that these are practically limited to Group A. Only 2 in Group B are well and working without thoracoplasty. An-

TABLE XII

PRESENT STATUS OF EMPYEMA IN THE 50 PATIENTS WHO ARE NOW WELL AND WORKING

	PNEUMO-THORAX	FLUID	REEXPANDED PARTIAL	REEXPANDED COMPLETE	OLEO-THORAX	THORACO-PLASTY
Group A: 42	7	4	3	13	8	7
Group B: 7	0	1	0	1	0	5
Uncertain: 1	0	0	1	0	0	0
Total	7	5	4	14	8	12

other interesting point is that in Group A, 42 out of 105 are well and working, whereas in Group B only 7 out of the 42 have been able to scale the heights to returned health.

SUMMARY

From this series we have tabulated certain facts and drawn certain conclusions.

Tuberculous empyema is a very common complication of artificial pneumothorax.

Our series comprises 154 patients with 64 known dead. There are 42 known mixed infections, and 47 recognized bronchopleural fistulas, of which 12 are not classified as having mixed infection.

The longer any series of empyema is followed, the higher will be the death rate. In our series, 140 have been followed three to eight years or longer.

The death rate is about 30 per cent in the uncontaminated tuberculous group and approximately twice that for the mixed infections and for the fistulas.

Recurrent serous effusions in therapeutic pneumothorax for tuberculosis, seropurulent effusions, and thick tuberculous pus are progressive stages of a tuberculous pleuritis. At any stage this may become secondarily infected or may rupture to the outside, either through a bronchus or through the chest wall, frequently after a period of years.

The problem of all empyemas is a dual one—composed of, first, the infection itself and, second, the abnormal pleural space. The attempt to control the former without obliteration of the latter is difficult, although sometimes possible.

About 70 per cent of the straight tuberculous group have survived the infection to date. Of these at least 30 per cent have ruptured or become secondarily infected. All of the remainder who still have fluid in the chest, either pus or oil, have that possibility ahead of them.

RESULTS OF TREATMENT

Aspiration Only: Of no value in Group B but in Group A, 40 per cent dead and 40 per cent well without further treatment.

Irrigation With Salt and Dyes: In Group A, 43 patients; 15 dead, 14 well, but only 5 without further treatment. In Group B, 33 patients; 19 dead, 5 well, but only 2 without further treatment. In Group B, however, salt and dye were frequently of great value in preparation for later thoracoplasty.

Oleothorax: Of no value in Group B but of great value in Group A where 11 of 22 patients are reported well without further treatment. Of these, 3 have completely obliterated the space; of the remainder, several have the possibility of perforation still ahead.

Tube or Thoracotomy: Contraindicated in Group A, frequently a necessity in Group B. Only one of 33 patients well without further treatment, 22 of the remainder are dead.

Thoracoplasty: Of great value in both Groups A and B; 16 cases in Group A, 12 in Group B. Total of 12 well and 7 dead. Of the 9 resting, 4 were operated upon within the last year. The better the patient's condition and the thinner the pleura, the better the end result. Many of the dead who have not had thoracoplasty probably passed through stages where thoracoplasty could have been done with minimal risk. In both groups, risk of thoracoplasty is less and chance of cure is more than with disease alone.

Of the 50 who are well and working, 14 are reexpanded, 12 have had thoracoplasty, 8 oleothorax, and the remaining 16 are divided between pneumothorax and pus with several partially reexpanded. In Group B only 7 of 42 patients are well and working. Of these, 5 have had thoracoplasty, one is reexpanded and one is continuing with pus in chest. We feel that our results can be improved.

SUGGESTIONS

Group A: Decide whether collapsed lung will probably be worth reexpanding later. In other words, is the chance of a good lung following reexpansion worth gambling for against the dangers of empyema? Even the reexpanded lungs in the presence of much disease or of prolonged fluid are frequently physiologically poor lungs. If the chance is not worth the gamble, and the patient is in fair condition and the opposite lung good, his chances of being alive and well are definitely greater with early thoracoplasty.

If the collapsed lung seems worth the gamble, attempt to keep the pleura dry with repeated aspirations; failing that, try oleothorax, hoping to aspirate that soon and continue with pneumothorax. If it cannot be controlled, try early thoracoplasty.

We know that of our 42 patients who had mixed infection empyemas, at least 16 of them passed through a stage, lasting for months or years, during which time the fluid contained only tubercle bacilli. In other words, more than one-third of them could have been spared the consequence of a mixed infection by obliteration of the pleural space during the earlier stage. Once secondary infection does occur,

vigorous treatment with the various dyes is valuable if treatment is begun soon after the infection develops and particularly if it is due to gram-positive cocci. In most instances it will be necessary to institute drainage; about one-half of the patients will survive their acute infection and will recover sufficiently to justify thoracoplasty being attempted.

DISCUSSION

DR. W. L. ROGERS, San Francisco.—I should like to make a transient comment on the twelve cases Dr. Woodruff passed over rather rapidly in which there was a demonstrated bronchial fistula, but in which they were unable to demonstrate a secondary infection. We have recently reviewed our statistics, and in a group of 20 patients with bronchial fistula, there were 7 in whom we never demonstrated at any time the evidence of a secondary infection. I think this is of interest because of the paper we heard previously at this meeting, and I do not believe the explanation given us is sufficient. In other words, several of these patients went from one to four years emptying the empyema by means of the bronchial tree, coughing up the empyema through the bronchial tree. That is a long period of time. I do not know the explanation, but I know such was the case in this group. Possibly it is because of the site of the bronchial fistula, or possibly it is because of the size. We also know that in this group, if the bronchial fistula is small, oleothorax works quite well and the oil may never be coughed up.

There is one case that I might comment on where I thought Dr. Eloesser showed his ingenuity. The patient was sent to me for a thoracoplasty. I was going right ahead, but Dr. Eloesser asked, "Why are you going to do a thoracoplasty?"

I was taken aback, and replied, "Well, I thought to get rid of the positive sputum and close a small empyema cavity."

"Where is the sputum coming from?" he said.

I replied, "I think it is coming from his lung."

"How do you know?" he asked.

I said, "Well, I don't know."

So we proceeded to find out. By injection dyes, lipiodol, etc., we were able to demonstrate that the patient was raising sputum from a small empyema pocket which was not secondarily infected and which contained tubercle bacilli; but between the attacks of raising the empyema he had no sputum, and what little phlegm he spat up was always negative. The days he did not cough up the dye, the sputum was invariably negative; and the days in which the dye appeared, his sputum was positive. The empyema pocket was filled with oil, which the patient has never spat up. Tubercle bacilli have been absent for one and a half years, and the patient is at work, quite well.

DR. LEO ELOESSER, San Francisco.—I do not remember whether in this case of which Dr. Rogers spoke the sputum was rendered negative by injection of the cavity or not.

DR. ROGERS.—Yes.

DR. ELOESSER.—I should like to call attention to a procedure that I think might possibly be tried. I have been interested in it for the last few years. It was devised for tuberculous empyema. As a matter of fact, its use has been disappointing in tuberculous empyema, but it has been of considerable value in nontuberculous empyema. It has also been of value, I think, in closing certain tuberculous empyemas. You will find it described in *Surgery, Gynecology, and Obstetrics* for June, 1935. I will pass around a little diagram that will show the operation better than I can describe it in words.

It consists of a skin flap which is sewed into the pleura so that it makes an opening into the chest by which pus can come out, but little or no air can enter. It obviates the use of a tube, which I think everybody will concede is a bad thing, especially in tuberculous empyema.

I shall pass the diagram around.

DR. H. BRODIE STEPHENS, San Francisco.—I believe I express the universal opinion of this group when I say we are grateful for this carefully prepared review by Dr. Woodruff. The author has given us an excellent study of a large group of cases, and from this study definite information for improvement in our present method of treatment of tuberculous empyema will result.

I would also like to say something about Dr. Eloesser's operation.* This very ingenious, single procedure has proved most valuable in the treatment of several cases of tuberculous empyema.

We all recognize from clinical experience that a tuberculous empyema, whether of the simple or mixed type, does better with closed drainage in comparison with an open method. A closed type of drainage is obtained by the use of the flap operation of Eloesser.

If one is dealing with a tuberculous empyema where the lung itself is not actually diseased, satisfactory drainage is obtained with the flap procedure and at the same time a slow expansion of the lung is brought about. The flap provides excellent drainage, too, in those cases where thoracoplasty is indicated. When a broncho-pleural fistula is present and persists even after thoracoplasty, the flap will act as a safety valve in preventing a recurrence of the empyema and, for this reason obviates the radical Shede operation.

*Surgery, Gynecology and Obstetrics, 60: 1096, 1097, June, 1935.

PLEURAL ENDOSCOPY AND PHOTOGRAPHY

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EVERYONE dealing more extensively with pleural endoscopy will soon realize that some considerable incongruity exists between the radiographic appearance and what is actually seen in the thoracoscope, in so far as pleural adhesions are concerned. Often enough great surprises are in store for the endoscopist. Adhesions believed to be made up of strands may prove to be membranes—the radiogram having only caught the free edges of the transverse membranes—and where membranes are expected a number of strands may give one instead, radiologically, the impression of one single coherent formation; or else it may be the lung tissue itself, overstretched by the pneumothorax treatment and directly adherent to the chest wall that gives the appearance of an adherent membrane. From a practical point of view, it is of importance to bear this incongruity in mind, particularly so when it comes to judging whether a case is a suitable one for cauterization or not. For some adhesions, which on the film look like surface adhesions and seem impossible to cauterize, can many times be burnt through entirely, or to a striking extent, whereas in other cases for which cauterization was believed to be an easy matter, this is contraindicated because the adhesions are found to be only slightly, or not at all, elastic and besides containing lung tissue to a great extent.

If on inspection of the radiogram, one is in doubt whether cauterization is feasible or not, one should never overlook to pass the thoracoscope to see how the land lies. Considering how enormously important Jacobaeus's method of cauterization of adhesions has proved to be in the collapse therapy, an omission in this respect may readily signify neglect. Experience would seem to have proved beyond doubt that it is often better to examine with the thoracoscope too much than too little, and that only endoscopy is able to give us a clear idea of existing conditions in the pleural space and to tell us whether cauterization of the adhesions is possible or not.

From an educational aspect and for the purpose of practical demonstration, the thoracoscopic picture is of definite interest. Even at an early stage of the history of endoscopy attempts were made to obtain photographic reproductions of what was observed in the thoracoscope. These attempts, however, either failed completely or proved of little success, the reason being that the source of light—the thoracoscopic lamp—was not of sufficient brilliancy. The beautiful pictures found in

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From the City of Stockholm Hospital for Tuberculosis, Söderby, Sweden.

atlases and textbooks illustrating Jacobaeus's operation are reproductions of drawings by hand and have nothing to do with direct photography of the pleural space. Kremer, however, in the autumn of 1934, published in Ztschr. f. Tuberk. photographs taken during cauterization. His instrumentarium consisted of a thoracoscope with a casette built in the direct optic axis.

In my own experiments beginning in April, 1934, I made use of a different method by employing a specially built camera by Kifa, Stockholm, coupled to the thoracoscope. It was not, however, until H. Kollmorgen collaborated with me and made the thoracoscope larger, enabling a greater quantity of light to enter the camera and larger lenses to be used, that the photographic experiments were met with success.

This new thoracoscope, of a diameter of 6 mm., has proved of exceedingly great value for cauterizing operations. It has also come into great use not only in Scandinavian countries but also elsewhere. Thanks to the strong light emitted, the thoracoscopic picture becomes exceedingly well defined and rich in contrasting details, particularly valuable in all those cases where the adhesions are such as to demand the greatest care in freeing the lung in order to avoid damaging the lung tissue. The perils associated with cauterization, which almost entirely refer to the damage that may be caused to the parenchyma, have thus been considerably reduced, due not only to the stronger light employed but also to the greater magnification. Complications have become less frequent. In the course of the last three years with close upon 400 cases of cauterization of adhesions, I have not met with a single case of that most dreaded complication, pleural empyema. The operation can be carried out with greater confidence and under a very different feeling of safety than formerly. The field of usefulness of cauterization has thus been enlarged. (In Nord. med. tidskr. and in Beitr. z. klin. d. Tuberk., 1935, an account will be found of the results attained by the experimental camera and the new optic instrument.)

The appearance of the special camera will be apparent from Fig. 1. It is built principally on the same lines as the experimental camera. As is evident from the above publications, a fastening device around the object glass enables the camera, by means of a simple manipulation, to be attached to the thoracoscope. For the proper adjustment of the picture the camera is provided with a side tube, corresponding to the finder in an ordinary camera. The optic system in the side tube is so arranged that it gives an erect image of the thoracoscopic picture. It is exceedingly simple, therefore, to adjust into the optic field any part of the pleural space one wishes to photograph.

The light is reflected into the side tube by a prism placed in the direct axis of the object glass of the camera and the ocular of the thoracoscope. On exposure the prism is automatically displaced sideways, thus enabling all the light emitted to enter the camera unhindered. As regards

the light itself, the device first published has been maintained. This device aimed at increasing the brilliancy of the thoracoscopic lamp by subjecting it to a momentous overcharge during the exposure by short circuiting a resistance enclosed in the photographic apparatus. The camera is also fitted with a compur shutter adjustable to exposures of one, one-half, and one-fifth seconds.

To obtain good pictures with the optic instrument concerned, it was necessary to use exposures of no less than one second. With this exposure, beautiful pictures were obtained in all cases where the lung could be brought to a standstill during the necessary period of exposure. If, on the other hand, it was impossible to arrest the lung movements, as often happens in operations on the left half of the chest where the wholly or partly compressed lung frequently moves synchronously with the cardiac action, photography was often without avail, or else unsatisfactory pictures due to blurring were obtained. If this drawback is to be avoided, the light must obviously be made still brighter in order to shorten the exposure. In experiments carried out in the course of the

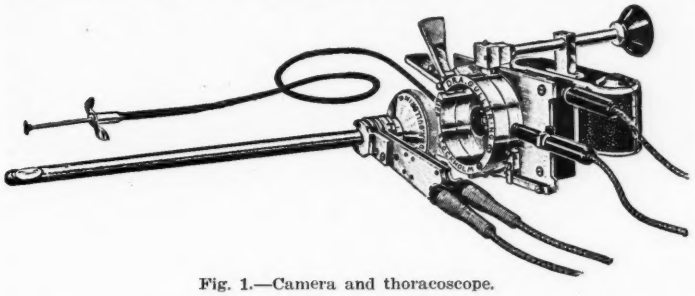


Fig. 1.—Camera and thoracoscope.

past twelve months with different thoracoscopes, it also proved of importance to obtain larger visual fields, thereby to enable the localization of lung and adhesions to be better demonstrated on the picture obtained.

Since the luminous power of the lamp could naturally be improved by shortening the optic system in the thoracoscope, a few instruments were made of different lengths. It then became clear that the best results were obtained by an optic system, 6.5 cm. shorter than that earlier employed. By using those kinds of glass for the lenses that still further diminished light absorption, a relative brilliancy was attained no less than five times as powerful as that obtained by the earlier thoracoscope. The visual field has been altered in the desired direction by reconstruction of the lenses; yet the larger visual field has been obtained at the expense of magnification.

Owing to the great luminosity of the new instrument, one has been able to reduce the exposure by about one-half. In nearly all cases, an exposure of half a second has, therefore, proved sufficient to obtain exceedingly sharp and well-defined photographs.

This thoracoscope, mainly intended for photography, is not suitable for cauterization in the same degree as the ordinary 6 mm. instrument. The magnification is, however, quite sufficient for all adhesions techni-

Fig. 2-*a*. Fig. 2-*b*. Fig. 2-*c*.

Fig. 3-*a*. Fig. 3-*b*. Fig. 3-*c*.

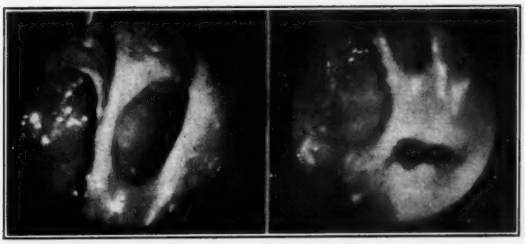
Figs. 2 and 3.—Two cases of cauterization of adhesions are shown in Fig. 2 *a*, *b*, *c*, and Fig. 3 *a*, *b*, *c*.

Fig. 4.—Shows to the right of the field an emphysematous vesicle, almost the size of a Brazil nut, visible after cauterization of a membranous adhesion, the free border of which is sharply defined.

cally of no special difficulty. In the case of widespread and short adhesions, on the other hand, where one easily runs the risk of damaging the lung tissue, the greatest possible magnification must naturally be resorted to.

Should one wish to follow such an operation photographically, it would be necessary to employ both instruments, which is a drawback of no great account, since both thoracoscopes are of the same caliber and fit into the same cannula. As a preliminary step, a general view is always taken with the short 6 mm. instrument; this is then exchanged for the long thoracoscope and cauterization is carried out. The operation completed, or before if it is wished to photograph some special phase of the procedure, the thoracoscope is again exchanged. The short instrument is introduced into the cannula, the camera connected, and further pictures taken.

In the photographic series taken, the individual pictures are qualitatively almost equivalent and very uniform. Failure of exposure is an



a. *b.*

Fig. 5.—The two pictures here are taken during a thoracoscopic examination of a cavity; *a*, depicts a trabecular design of a cavity in the upper lobe, and *b*, a draining bronchus cauterized.

exception, and the method is, as now elaborated, ready for use in endoscopy.

Quite recently an attempt was made to use a colored film, Kodachrome, Type A. Beautiful pictures were obtained with an exposure of one second. The color design obtained is mainly made up of various shades of red, brown, yellow white and blue black, which colors accord well with those present in the thoracoscopic picture. The picture is sharply defined and is rich in details. It reproduces in an excellent manner the display of brilliant coloring existing in the pleural space, particularly in evidence in hyperemic parts of the pleura, and where adhesions are more vascular.

STUDIES OF THE CARDIAC OUTPUT OF ANESTHETIZED DOGS DURING CONTINUOUS AND INTERMITTENT INFLATION OF THE LUNGS*

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CIRCULATORY failure is a not infrequent complication during or after major operations on the chest. It is our belief that the degree of failure is often out of proportion to the degree of operative shock which might be expected from the extent of trauma and blood loss. Variations from the normal state which might influence the circulation during a major operation on the chest include: (1) the collapse of more or less lung; (2) the ligation of one or more branches of the pulmonary artery; (3) the compression of the heart and its great vessels; (4) the compression of the intrapulmonary vessels if the lungs are inflated artificially.

It has already been shown experimentally that the first of these changes is regularly accompanied by a decrease in the minute volume output of the heart.[1, 2] It has been shown further that sudden occlusion of either primary branch of the pulmonary artery does not always influence the cardiac output.[3] More recently a drop in cardiac output was observed when the chest of an animal was opened wide and respiration maintained by intermittent insufflation of the lungs.[4] The abnormal conditions under which these latter experiments were performed did not involve the collapse of pulmonary tissue and were not accompanied by ligation of any of the branches of the pulmonary artery. The heart and its afferent vessels, however, were exposed to a change in pressure, and the artificial ventilation, which was used necessarily, caused an increase in the pressure within the air passages of the lungs.

The following experiments were undertaken in order to study more completely the effects of intratracheal insufflation under varying degrees of positive pressure on the minute volume output of the heart. When the experiments were planned, it was thought that that part of the tamponade effect resulting from distention of the lungs could be avoided by opening the chest widely and allowing the lungs to bulge through the opening. Hence studies were made after wide-open thoracotomy for comparison with those that were made on the intact animals. It soon became obvious, however, that when the lungs were distended at high pressures, compression of the heart was not avoided by opening the chest.

*From the Surgical Pathology Laboratory of the College of Physicians and Surgeons, Columbia University, and the Department of Surgery, the Presbyterian Hospital, New York.

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Hence the effects of cardiac compression and compression of the intrapulmonary vessels were not studied separately (Fig. 1).

Dogs were used. They were anesthetized by the intravenous injection of barbital sodium and breathed oxygen throughout the period of experimentation. Most of the studies were made during continuous inflation, because it was found that the intratracheal pressure could be more accurately controlled when there was no lung motion. In some of the intact animals, however, measurements of cardiac output were made during periods of intermittent inflation for comparison with those previously

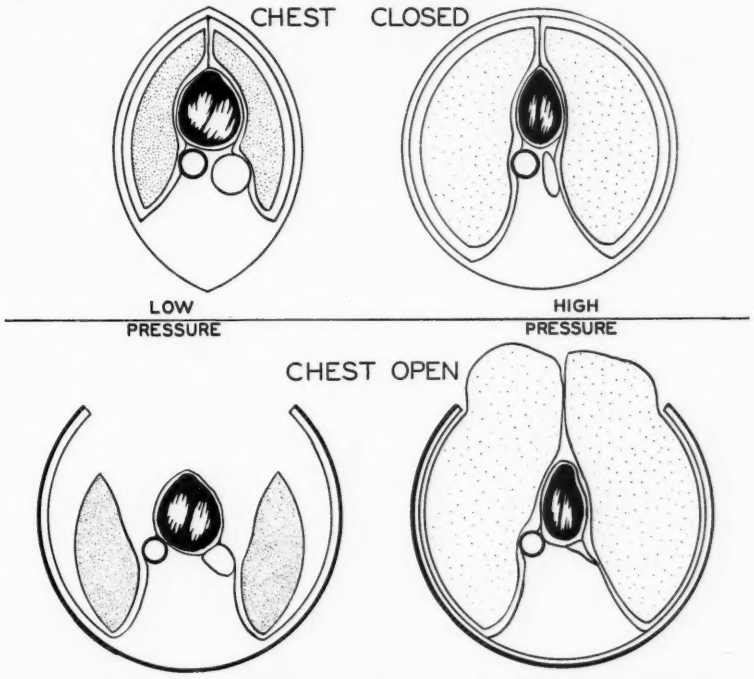

Fig. 1.—A diagram to show the effects on the heart and great vessels of the various experimental conditions. There was least compression with chest closed and low pressure; slightly more with chest open and low pressure; and considerable compression with high pressure, whether the chest was open or closed.

reported following open thoracotomy. Previous studies[2, 4] have convinced us that lung motion in itself does not have any significant effect in propelling the blood.

THE EFFECTS OF CONTINUOUS INFLATION AT HIGH PRESSURES WITH CHEST CLOSED

Method.—In four experiments the cardiac outputs during periods of natural breathing were compared with those during a period in which the lungs were inflated at pressures varying between 20 and 30 mm. of mercury.

The apparatus used was identical with that described in a previous paper[4] and is illustrated in the accompanying diagram (Fig. 2). When anesthesia was complete, the intratracheal cannula was inserted, fastened securely in place with two ligatures, and the animals connected with the closed respiratory system. Oxygen absorption could then be computed from measurements of the kymographic tracings. Arterial and venous blood samples were obtained, the former through a cannula inserted into the femoral artery, and the latter by means of a special cannula[5] inserted into the right heart through a slit in the external jugular vein. The oxygen contents of the blood samples were estimated at once accord-

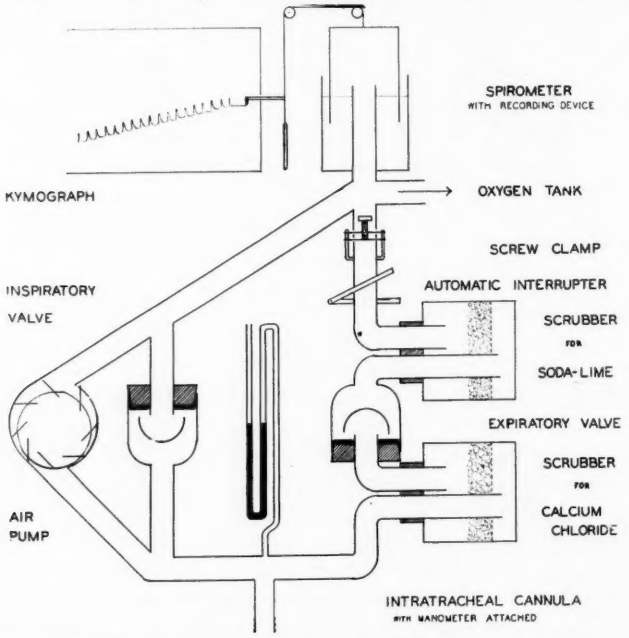

Fig. 2.—A diagram of the apparatus used in the first four experiments. In the remaining experiments rubber flutter valves were used in place of the mercury seal valves, and the calcium chloride scrubber was not used. Inspiration may take place either through the valve or by means of the pump; partial occlusion of the expiratory airway with screw clamp or automatic interrupter with the pump going causes continuous or intermittent elevation of intratracheal pressure.

ing to the manometric method of Van Slyke and Neill[6] and the cardiac output calculated according to the Fick formula[7]:

$$\frac{\text{c.c. of oxygen absorbed per minute}}{\text{amount of oxygen absorbed by 1 c.c. of blood in passing through the lungs}} = \text{c.c. of blood passing through the lungs per minute}$$

A preliminary period was studied with the dog breathing naturally through the inspiratory and expiratory valves. The air pump was then

started, the inspiratory valve clamped off, and the intratracheal pressure raised by obstructing the expiratory tube by means of the screw clamp, until the desired pressure was reached. A second set of observations was made immediately. The intratracheal pressure was then released and the dog allowed to breathe naturally again through the valves while a final period was studied.

Results.—In three of the four experiments (Nos. 93, 94, and 108) cardiac output showed a pronounced decrease; in the fourth (No. 96) the decrease was significant, but not as great. When natural breathing was resumed, the cardiac output returned toward the control level in all of the experiments, in two instances (Nos. 93 and 108) to slightly less than the control, and in the other two (Nos. 94 and 96) to a higher figure than the control. The data of these experiments are summarized in Table I.

THE EFFECTS OF CONTINUOUS INFLATION AT HIGH AND LOW PRESSURES WITH THE CHEST OPEN

Method.—In seven experiments the cardiac outputs were determined, first during a control period of natural breathing with the chest closed, and later, during several periods of continuous inflation, with the chest widely opened.

The experimental conditions and the apparatus were essentially the same as in the previous group, differing only in the substitution of flutter valves for the mercury seal valves and the elimination of the calcium chloride scrubber. When the control studies were completed, the pump was started and the intratracheal pressure raised very slightly by means of the screw clamp on the expiratory tube. Then the chest was opened by splitting the sternum in the midline, and the ribs were held wide apart with a self-retaining retractor. After this, the cardiac output studies were repeated: (1) with the intratracheal pressure so adjusted that the lungs did not fill the chest; (2) at increased pressures so that the lungs just filled the chest, and (3) at pressures that caused the lungs to bulge through the thoracotomy wound. The order in which these studies were made was not always the same, but in every case at least twenty minutes were allowed to elapse after changing the intratracheal pressure before the next determination of cardiac output was made. This was done in order to allow sufficient time for the experimental conditions to have an effect.

Results.—In five of the seven experiments (Nos. 132, 134, 136, 137, and 138) there was a fall in cardiac output during the period in which the lungs did not fill the chest. In the other two experiments (Nos. 133 and 135) cardiac output rose, the rise being slight in one instance (No. 135) and pronounced in the other (No. 133). When the distention of

TABLE I

THE EFFECTS OF CONTINUOUS INFLATION AT HIGH PRESSURES IN NORMAL DOGS

	SUMMARY OF EXPERIMENTAL DATA	CONTROL PERIOD	CONTINUOUS INFLATION	NATURAL BREATHING
Experiment 93	Intratracheal pressure (mm. mercury)		20	
April 17, 1934	Pulse rate	128	(not palpable)	156
Weight 11.3 kg.	Oxygen content of arterial blood (per cent by volume)	20.1	24.0	23.9
Female	Oxygen content of venous blood (per cent by volume)	16.5	16.9	20.1
Barb. sod. 3.4 gm.	Arteriovenous oxygen difference	3.6	7.1	3.8
	Oxygen absorbed (c.c. per minute)	64.0	32.4	58.4
	Volume output of blood from the heart (c.c. per minute)	*1778*	*457*	*1539*
	Change in cardiac output (per cent)		−74.3	−13.4
Experiment 94	Intratracheal pressure (mm. mercury)		30	
April 24, 1934	Pulse rate	150	152	186
Weight 12.9 kg.	Oxygen content of arterial blood (per cent by volume)	22.9	23.6	21.6
Male	Oxygen content of venous blood (per cent by volume)	21.1	18.1	19.7
Barb. sod. 3.9 gm.	Arteriovenous oxygen difference	1.8	5.5	1.9
	Oxygen absorbed (c.c. per minute)	88.9	34.3	112.2
	Volume output of blood from the heart (c.c. per minute)	*4941*	*624*	*5906*
	Change in cardiac output (per cent)		−87.3	+19.5
Experiment 96	Intratracheal pressure (mm. mercury)		20	
May 16, 1934	Pulse rate	142	144	136
Weight 14.5 kg.	Oxygen content of arterial blood (per cent by volume)	19.9	20.6	19.9
Male	Oxygen content of venous blood (per cent by volume)	16.3	15.6	17.9
Barb. sod. 4.4 gm.	Arteriovenous oxygen difference	3.6	5.0	2.0
	Oxygen absorbed (c.c. per minute)	69.8	79.1	51.2
	Volume output of blood from the heart (c.c. per minute)	*1940*	*1583*	*2562*
	Change in cardiac output (per cent)		−18.4	+32.0
Experiment 108	Intratracheal pressure (mm. mercury)		26	
March 29, 1935	Pulse rate	140	120	126
Weight 14.9 kg.	Oxygen content of arterial blood (per cent by volume)	21.0	23.4	22.3
Male	Oxygen content of venous blood (per cent by volume)	18.1	18.9	19.0
Barb. sod. 4.5 gm.	Arteriovenous oxygen difference	2.9	4.5	3.3
	Oxygen absorbed (c.c. per minute)	68.9	39.3	73.7
	Volume output of blood from the heart (c.c. per minute)	*2376*	*874*	*2236*
	Change in cardiac output (per cent)		−63.2	−5.9

the lungs was increased so that they just filled the chest, in two instances (Nos. 133 and 134) the cardiac output dropped to a level lower than that which it maintained during the preceding period of less inflation. In the other experiment (No. 132) it was a little higher than it was in the preceding period. In all of the experiments during the period of high intratracheal pressure, when the distended lungs protruded from the chest, the cardiac output was lower than in the periods of less inflation. The data of these experiments are summarized in Table II.

THE EFFECTS OF INTERMITTENT AND CONTINUOUS INFLATION AT LOW PRESSURES WITH THE CHEST CLOSED

Method.—In nine experiments the cardiac outputs during a control period of normal breathing were compared with those during periods in which respiratory paralysis was produced with curare, and the animals were insufflated intermittently and continuously.

The experimental conditions and apparatus were the same as in the previous group. Cardiac output was first determined with the dog breathing naturally, then 2 c.c. of a 5 per cent solution of curare syrup* was injected through the cannula in the femoral artery. If respirations did not cease within five minutes, 1 c.c. more was added, and in a few instances a fourth or fifth cubic centimeter was necessary. As soon as respirations had ceased, artificial ventilation was begun, in all experiments but one beginning with intermittent inflation so regulated that the maximum intratracheal pressures varied between 4 and 7 mm. of mercury. Intermittent inflation was continued for at least twenty minutes before the next determination of cardiac output was made. Insufflation was then made continuous at pressures varying between 3 and 4 mm. of mercury, and after another interval of at least twenty minutes the determination of cardiac output was repeated. In six of these experiments studies of cardiac output during a second period of intermittent inflation were made. In the final experiment the observations during continuous inflation were made immediately after respirations had ceased and just preceding a single period of intermittent inflation.

Results.—In six of the eight experiments (Nos. 113, 116, 117, 121, 123, and 124) in which curarization was followed by intermittent inflation, there was a significant drop in cardiac output. The other two (Nos. 114 and 115) showed a slight fall and a slight rise, respectively. After changing to continuous inflation at lower pressures, there was a rise in cardiac output in every instance. In four of the eight experiments (Nos. 113, 114, 115, and 117) cardiac output rose to a figure exceeding the control level. In the second period of intermittent inflation, obtained in six instances (Nos. 113, 114, 115, 117, 121, and 124), the output again

*One gram of crude curare syrup (Eimer and Amend) dissolved in 20 c.c. of distilled water and filtered.

TABLE II

THE EFFECTS OF CONTINUOUS INFLATION AT LOW AND HIGH PRESSURES WITH THE CHEST OPEN

		CONTROL PERIOD NATURAL BREATHING	CHEST OPEN LUNGS DO NOT FILL CHEST	CHEST OPEN LUNGS FILL CHEST	CHEST OPEN LUNGS BULGE FROM CHEST
Experiment 132	Intratracheal pressure (cm. of water)		4	10	20
Nov. 25, 1936	Pulse rate	98	116	110	120
Weight 9.7 kg.	Oxygen content of arterial blood (per cent by volume)	18.4	17.5	17.6	(17.5)
Male	Oxygen content of venous blood (per cent by volume)	15.1	12.8	13.7	9.3
Barb. Sod. 3.0 gm.	Arteriovenous oxygen difference	3.3	4.7	3.9	8.2
	Oxygen absorbed (c.c. per minute)	56.1	48.8	50.8	50.8
	Volume output of blood from the heart (c.c. per minute)	*1700*	*1038*	*1304*	*620*
	Change in cardiac output (per cent)		−39.0	−23.3	−63.5
Experiment 133	Intratracheal pressure (cm. of water)		10	16	20
Dec. 1, 1936	Pulse rate	156	134	114	116
Weight 8.0 kg.	Oxygen content of arterial blood (per cent by volume)	24.0	23.3	23.7	24.4
Female	Oxygen content of venous blood (per cent by volume)	18.9	20.5	19.9	18.5
Barb. Sod. 2.5 gm.	Arteriovenous oxygen difference	5.1	2.8	3.8	5.9
	Oxygen absorbed (c.c. per minute)	50.0	60.4	60.4	58.8
	Volume output of blood from the heart (c.c. per minute)	*981*	*2157*	*1590*	*996*
	Change in cardiac output (per cent)		+119.8	+62.0	+1.5
Experiment 134	Intratracheal pressure (cm. of water)		4	7	20
Dec. 8, 1936	Pulse rate	154	90	108	104
Weight 14.5 kg.	Oxygen content of arterial blood (per cent by volume)	23.0	24.0	23.7	24.0
Female	Oxygen content of venous blood (per cent by volume)	20.0	20.6	19.8	19.2
Barb. Sod. 4.4 gm.	Arteriovenous oxygen difference	3.0	3.4	3.9	4.8
	Oxygen absorbed (c.c. per minute)	56.3	(56.3)	(56.3)	(56.3)
	Volume output of blood from the heart (c.c. per minute)	*1878*	*1658*	*1444*	*1173*
	Change in cardiac output (per cent)		−11.6	−23.0	−37.5

TABLE II—CONT'D

Experiment 135	Intratracheal pressure (cm. of water)		7	28
Jan. 13, 1937	Pulse rate	108	106	96
Weight 17.6 kg.	Oxygen content of arterial blood (per cent by volume)	17.7	18.7	21.3
Female	Oxygen content of venous blood (per cent by volume)	13.6	14.7	15.6
Barb. Sod. 5.3 gm.	Arteriovenous oxygen difference	4.1	4.0	5.7
	Oxygen absorbed (c.c. per minute)	72.7	76.8	85.0
	Volume output of blood from the heart (c.c. per minute)	*1774*	*1921*	*1490*
	Change in cardiac output (per cent)		+8.2	−16.0
Experiment 136	Intratracheal pressure (cm. of water)		6.5	25
Jan. 23, 1937	Pulse rate	150	98	106
Weight 16.4 kg.	Oxygen content of arterial blood (per cent by volume)	22.3	22.8	22.6
Female	Oxygen content of venous blood (per cent by volume)	17.7	17.3	11.2
Barb. Sod. 5.0 gm.	Arteriovenous oxygen difference	4.6	5.5	11.4
	Oxygen absorbed (c.c. per minute)	79.1	88.5	92.8
	Volume output of blood from the heart (c.c. per minute)	*1720*	*1608*	*814*
	Change in cardiac output (per cent)		−6.4	−52.6
Experiment 137	Intratracheal pressure (cm. of water)		4.5	19
Feb. 17, 1937	Pulse rate	160	112	108
Weight 12.3 kg.	Oxygen content of arterial blood (per cent by volume)	20.7	21.3	22.1
Female	Oxygen content of venous blood (per cent by volume)	18.4	18.4	18.9
Barb. Sod. 3.7 gm.	Arteriovenous oxygen difference	2.3	2.9	3.2
	Oxygen absorbed (c.c. per minute)	91.7	86.5	73.0
	Volume output of blood from the heart (c.c. per minute)	*3988*	*2982*	*2281*
	Change in cardiac output (per cent)		−25.2	−42.8
Experiment 138	Intratracheal pressure (cm. of water)		4	28
Feb. 20, 1937	Pulse rate	132	108	112
Weight 23.0 kg.	Oxygen content of arterial blood (per cent by volume)	22.7	23.4	25.9
Female	Oxygen content of venous blood (per cent by volume)	19.1	19.2	14.4
Barb. Sod. 7.0 gm.	Arteriovenous oxygen difference	3.6	4.2	11.5
	Oxygen absorbed (c.c. per minute)	99.9	93.9	82.2
	Volume output of blood from the heart (c.c. per minute)	*2774*	*2236*	*715*
	Change in cardiac output (per cent)		−19.0	−74.5

TABLE III

THE EFFECTS OF INTERMITTENT AND CONTINUOUS INFLATION AT LOW PRESSURES IN CURARIZED DOGS

	SUMMARY OF EXPERIMENTAL DATA	CONTROL PERIOD NATURAL BREATHING	INTERMITTENT INFLATION	CONTINUOUS INFLATION	INTERMITTENT INFLATION
Experiment 113	Intratracheal pressure (mm. mercury)		6 – 3	4	8 – 5
Nov. 13, 1935	Pulse rate	168	150	120	125
Weight 19.0 kg.	Oxygen content of arterial blood (per cent by volume)	18.6	18.5	20.3	21.7
Male	Oxygen content of venous blood (per cent by volume)	16.1	15.4	19.2	15.6
Barb. sod. 6.7 gm.	Arteriovenous oxygen difference	2.5	3.1	1.1	6.1
	Oxygen absorbed (c.c. per minute)	101.3	72.2	104.2	95.9
	Volume output of blood from the heart (c.c. per minute)	*4053*	*2328*	*9470*	*1564*
	Change in cardiac output (per cent)		–42.5	+133.6	–61.3
Experiment 114	Intratracheal pressure (mm. mercury)		6 – 0	3	6 – 0
Nov. 20, 1935	Pulse rate	120	110	100	112
Weight 7.5 kg.	Oxygen content of arterial blood (per cent by volume)	15.3	17.4	16.7	17.6
Female	Oxygen content of venous blood (per cent by volume)	13.5	13.0	14.9	11.1
Barb. sod. 2.3 gm.	Arteriovenous oxygen difference	1.8	4.4	1.8	6.5
	Oxygen absorbed (c.c. per minute)	51.6	111.2	64.8	95.1
	Volume output of blood from the heart (c.c. per minute)	*2865*	*2526*	*3602*	*1463*
	Change in cardiac output (per cent)		–11.8	+25.9	–48.9
Experiment 115	Intratracheal pressure (mm. mercury)		7 – 1	3	5 – 1
Nov. 27, 1935	Pulse rate	140	128	140	133
Weight 10.1 kg.	Oxygen content of arterial blood (per cent by volume)	14.4	16.3	16.8	16.5
Male	Oxygen content of venous blood (per cent by volume)	10.5	13.9	14.8	13.9
Barb. sod. 3.0 gm.	Arteriovenous oxygen difference	3.9	2.4	2.0	2.6
	Oxygen absorbed (c.c. per minute)	64.1	42.8	36.5	31.1
	Volume output of blood from the heart (c.c. per minute)	*1644*	*1785*	*1827*	*1204*
	Change in cardiac output (per cent)		+8.5	+11.1	–26.7
Experiment 116	Intratracheal pressure (mm. mercury)		4 – 2		
Dec. 17, 1935	Pulse rate	126	122	162	
Weight 9.7 kg.	Oxygen content of arterial blood (per cent by volume)	20.9	20.6	21.7	
Female	Oxygen content of venous blood (per cent by volume)	19.3	16.6	20.4	
Barb. sod. 3.0 gm.	Arteriovenous oxygen difference	1.8	4.0	1.3	
	Oxygen absorbed (c.c. per minute)	41.7	43.5	35.3	
	Volume output of blood from the heart (c.c. per minute)	*2315*	*1088*	*2713*	
	Change in cardiac output (per cent)		–53.0	+17.1	

TABLE III—CONT'D

Experiment 117	Intratracheal pressure (mm. mercury)		6 – 2	3	6 – 2
Jan. 3, 1936	Pulse rate	96	115	80	108
Weight 10.8 kg.	Oxygen content of arterial blood (per cent by volume)	21.1	19.7	21.7	20.4
Female	Oxygen content of venous blood (per cent by volume)	20.0	16.6	20.6	18.9
Barb. sod. 3.3 gm.	Arteriovenous oxygen difference	1.1	3.1	1.1	1.5
	Oxygen absorbed (c.c. per minute)	53.3	43.8	39.5	41.3
	Volume output of blood from the heart (c.c. per minute)	*4846*	*1412*	*3587*	*2755*
	Change in cardiac output (per cent)		−70.7	−25.8	−45.2
Experiment 121	Intratracheal pressure (mm. mercury)		5 – 3	4	4 – 3
Feb. 7, 1936	Pulse rate	154	160	80	160
Weight 11.8 kg.	Oxygen content of arterial blood (per cent by volume)	16.8	17.4	18.0	17.1
Female	Oxygen content of venous blood (per cent by volume)	14.1	7.7	14.5	7.4
Barb. sod. 3.5 gm.	Arteriovenous oxygen difference	2.7	9.7	3.5	9.7
	Oxygen absorbed (c.c. per minute)	60.3	46.2	32.5	50.4
	Volume output of blood from the heart (c.c. per minute)	*2233*	*476*	*928*	*519*
	Change in cardiac output (per cent)		−78.7	−58.4	−76.2
Experiment 123	Intratracheal pressure (mm. mercury)		7 – 2	3	
Feb. 21, 1936	Pulse rate	144	168	128	
Weight 13.9 kg.	Oxygen content of arterial blood (per cent by volume)	23.1	22.1	22.6	
Male	Oxygen content of venous blood (per cent by volume)	20.6	16.4	19.3	
Barb. sod. 4.2 gm.	Arteriovenous oxygen difference	2.5	5.7	3.3	
	Oxygen absorbed (c.c. per minute)	78.7	79.5	60.0	
	Volume output of blood from the heart (c.c. per minute)	*3147*	*1394*	*1880*	
	Change in cardiac output (per cent)		−55.7	−40.2	
Experiment 124	Intratracheal pressure (mm. mercury)		7 – 2	4	7 – 2
March 6, 1936	Pulse rate	180	204	144	160
Weight 12.6 kg.	Oxygen content of arterial blood (per cent by volume)	16.3	19.0	20.2	20.4
Male	Oxygen content of venous blood (per cent by volume)	13.9	13.5	17.4	11.9
Barb. sod. 3.8 gm.	Arteriovenous oxygen difference	2.4	5.5	2.8	8.5
	Oxygen absorbed (c.c. per minute)	86.6	85.0	62.0	59.0
	Volume output of blood from the heart (c.c. per minute)	*3608*	*1545*	*2216*	*694*
	Change in cardiac output (per cent)		−56.8	−38.5	−80.7
Experiment 126	Intratracheal pressure (mm. mercury)			4	10 – 2
March 20, 1936	Pulse rate	162		120	180
Weight 13.2 kg.	Oxygen content of arterial blood (per cent by volume)	21.4		24.8	22.8
Female	Oxygen content of venous blood (per cent by volume)	19.9		24.2	19.6
Barb. sod. 4.0 gm.	Arteriovenous oxygen difference	1.5		0.6	3.2
	Oxygen absorbed (c.c. per minute)	62.4		58.8	59.0
	Volume output of blood from the heart (c.c. per minute)	*4160*		*9797*	*1844*
	Change in cardiac output (per cent)			+133.0	−55.6

fell; in four (Nos. 113, 114, 115, and 124) to a figure below that of the previous similar period. In the last experiment (No. 126) in which the observations during continuous inflation were made immediately after respiratory paralysis had set in, cardiac output rose well above the control figure. After changing to intermittent inflation, the output was found to have fallen to below the control. The data of these experiments are summarized in Table III.

DISCUSSION

To distinguish absolutely between the effects of compression of the heart in hindering its filling and the effects of compression of the intrapulmonary vessels in hindering its emptying is not possible on the basis of these experiments. Nevertheless, certain inferences may be drawn. In the first four experiments, the high intratracheal pressure in a closed chest must have been transmitted to all of the intrathoracic viscera, greatly reducing the filling of the right heart by pressure upon it and its afferent vessels. A comparable effect on the cardiac output was also observed when high pressures were used with the chest open. In these experiments the lungs bulged from the chest wound, meeting in the midline over the heart, and squeezing it between them. Regardless of the mechanism whereby the effect was produced, these experiments demonstrate very clearly that inflation of the lungs at high pressures had a pronounced effect on the circulation whether the chest was open or closed. Previous investigators[8-10] have shown that the heart is capable of overcoming obstruction of the pulmonary vessels to a great extent. It therefore seems likely that in these experiments compression of the heart and its afferent vessels had a greater effect than compression of the intrapulmonary vessels.

When the intratracheal pressure was kept low during continuous inflation with the chest closed, the expansion of the lungs was maintained only by the presence of an intact chest wall, as shown by the fact that when the same pressures were used with the chest open the lungs failed to fill the chest. Thus, the effect of the elastic recoil of the lungs was still present, some intrathoracic "negative" pressure remained, and no compression of the heart existed. When the chest was open, however, the effect of the elastic recoil of the lungs in producing a "negative" pressure in the chest was eliminated, and some compression was present due to the effect of atmospheric pressure on the heart and great vessels. If it is true that the circulation is much sooner affected by compression of the heart than by compression of the intrapulmonary vessels, there should have been a greater tendency for the cardiac output to fall when the chest was open than when it was closed. That this was the case is suggested by the fact that when the chest was open, cardiac output was lower than the control reading (chest closed, natural breathing) in five

experiments out of seven; whereas when the chest was closed, the readings were lower than the control readings in only four out of nine instances.

To produce intermittent inflation of the lungs in the curarized animals, it was necessary to raise the intratracheal pressure rhythmically to a higher figure than was used during continuous inflation, and so a higher mean pressure resulted. Under these conditions there was a consistent and significant decrease in cardiac output, both when the chest was open and when the chest was closed. The observations which were made during intermittent inflation in the open-chest preparation have already been reported,[4] and the decrease in cardiac output was attributed solely to the loss of intrathoracic "negative" pressure. In the light of our more recent observations, however, that open thoracotomy sometimes is unassociated with a fall in cardiac output when inflation of the lungs is controlled so that they do not fill the chest, this does not appear to be the true explanation. It seems more logical to attribute the decrease in cardiac output which was observed, both with chest closed and open, to compression of the heart and its afferent vessels.

These observations on animals seemed to be of sufficient interest to justify the time that they required because they give some conception of the mechanism by which positive inflation of the lungs may have a harmful effect on the circulation which, under certain conditions, may be directly responsible for a sudden death. The experiments suggest that the danger of inflating the lungs, either when the chest is closed or when the chest is open, is primarily due to a compression effect on the heart which interferes with its filling and not to pressure on the intrapulmonary vessels which creates a resistance against which the right heart must empty. The increasing frequency with which operative procedures of great magnitude are being carried out within the thoracic cavity has necessitated the frequent use of positive insufflation of the lungs. In order that the anesthetist may control the degree of intratracheal pressure, modern anesthesia machines are equipped with manometers. In the final analysis, however, the intratracheal pressure in itself may not be the important factor at all. The important factor probably is the effect of distended lungs in pressing on the heart and great vessels. In the absence of a means for recording this compression effect of the lungs, it is of the utmost importance for the anesthetist to realize that the lowest intratracheal pressure which will maintain ventilation is the safest. When a patient is in shock with the venous return to the heart already reduced, a compression effect which under normal conditions would be tolerated might lead to sudden death.

Although we have no means of measuring directly the pressure that is being exerted on the heart and great vessels, it may be that this can

be arrived at indirectly by recording venous pressures. Studies of venous pressure changes in relation to experimental variations of intrathoracic pressure are now in progress.

SUMMARY

Twenty experiments on anesthetized dogs are reported. In a group of four dogs the cardiac output during normal respiration was contrasted with that during a period in which the lungs were inflated at pressures varying between 20 and 30 mm. of mercury. In a group of seven dogs cardiac output during normal respiration was compared to that following open thoracotomy and continuous pulmonary inflation at both high and low pressures. In a group of nine dogs cardiac output during normal respiration was compared with that following curarization and intermittent or continuous inflation of the lungs at low pressures. It was found that:

1. High intratracheal pressure in intact dogs was followed in every instance by a significant fall in cardiac output.

2. Following open thoracotomy and continuous inflation at low pressures, there was a fall in cardiac output in five cases; in one case there was a slight rise in output, and in one there was a pronounced rise. When continuous inflation was continued at higher pressures, there were corresponding decreases in cardiac output.

3. Following respiratory arrest due to curare and intermittent inflation of the lungs, the cardiac output fell significantly in six of eight cases; in two the change was small. When intermittent inflation was replaced by continuous at lower pressures, there was an increase in cardiac output in every instance.

It is thought that these changes in cardiac output are more directly related to the pressure to which the heart and great vessels are subjected, than to the changes in intraalveolar pressure. It is, therefore, suggested that the chief danger of pulmonary insufflation is an indirect effect which hinders the filling of the heart, rather than a direct effect on the flow of blood through the lungs.

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CONTINUOUS ANTERIOR SIPHON DRAINAGE AND IRRIGATION OF PYOPNEUMOTHORAX*

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IN CERTAIN cases of pyopneumothorax, periodic drainage of the pleural sac by thoracentesis is inadequate and drainage by means of a tube anchored in the chest wall must be used instead. For guidance in the selection of these cases, the criteria described by Riggins and Amberson[1] are recommended.

The method of obtaining continuous tube drainage that is commonly used in cases of simple pyothorax is often employed in cases of pyopneumothorax. This method consists of the introduction of a tube of large caliber into the pleural sac by resecting part of a rib low in the posterolateral portion of the hemithorax. In pyopneumothoraces, the pneumothorax and the tuberculosis of the lung and pleura that are present result in behavior which differs from that of simple pyothoraces, in which the etiology is usually nontuberculous, and the drainage of a pyopneumothorax through the conventional posterior wound is unsatisfactory. Consequently, we employ the method of drainage illustrated in the accompanying diagram.

The flow of irrigating liquid from the liter flask, *10*, is controlled by a screw clamp, *11*, and a Murphy drip bulb, *12*. In the irrigation line, *13*, the liquid is warmed by some type of heating unit, *14*, before it passes through a soft rubber catheter, *15*, into the pleural sac, *16*. The pint bottle, *17*, acts as a trap which permits air to enter the pleural sac via the irrigation line, so as to equalize with the atmospheric pressure any changes which may occur in the intrapleural pressure. The clamp, *18*, can be used to disconnect this trap if subatmospheric intrapleural pressure is desired to promote reexpansion of the lung after the bronchopleural fistula has closed.

The intrapleural end of the drainage tube, *19*, which is shown in detail in the inset (Fig. 1), is held in place by an elbow of aluminum tubing, *20*, placed within that part of the drainage tube which is anchored in the chest wall. Liquid is drawn from the pleural sac through the drainage line, *21*, into the pint bottle, *22*, which serves to maintain the action of the siphon and to prevent aspiration of evacuated liquid back into the pleural sac. From this bottle, *22*, the liquid passes through a tube which opens underneath liquid in the gallon bottle on the floor.

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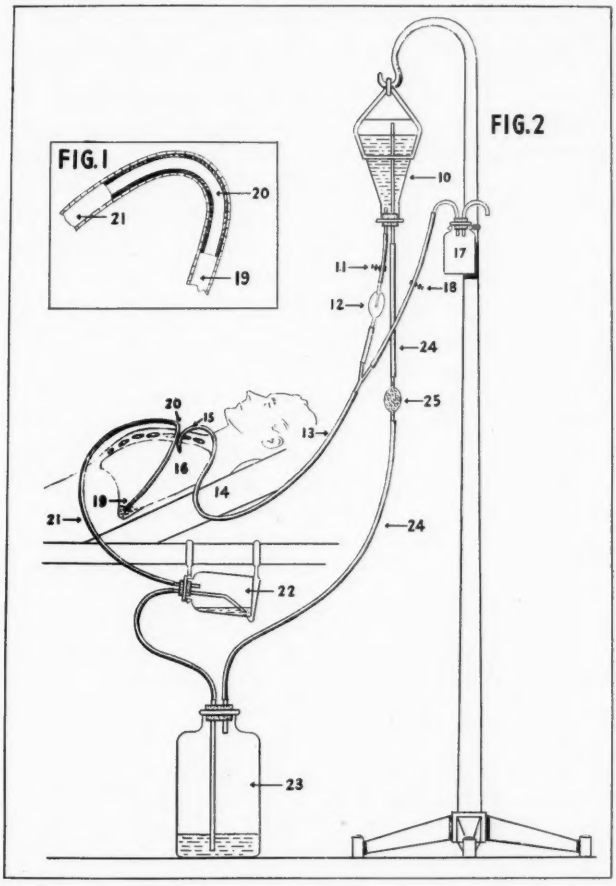
The partial vacuum created in the bottom of the irrigating flask, *10*, may be utilized to aid the action of the siphon by connecting the partial vacuum to the effluent bottle, *23*, by means of a line of tubing, *24*, into which a gauze filter, *25*, has been introduced.

Soft rubber tubing with a thick wall and an inside diameter of 5 mm. is used for all connecting lines. The glass tubing used is of 7 mm.



outside diameter. The drainage tube, *19*, is of soft rubber with a thin wall and is of 5 mm. inside diameter. The elbow of aluminum tubing* has a wall 1 mm. in thickness and an outside diameter of 5 mm.

Anterior siphon drainage has been found to have the following advantages over posterior drainage: (1) More exact control of the intrapleural pressure is possible. (2) Leakage of pus and irrigating liquid

*This tubing can be obtained from Patterson Bros., 27 Park Row, New York City.

around the drainage tube is prevented. (3) There is less discomfort for the patient. (4) Maintenance of drainage and irrigation is more convenient for the nurse and physician.

It must be emphasized that the only claim made for anterior irrigation and siphon drainage is that the method is more efficient than posterior drainage in cases of pyopneumothorax. The chief objective of tube drainage of the pleural sac in cases of tuberculous pyopneumothorax, complicated by secondary infection and persistent bronchopleural fistula, remains the preparation of the patient for thoracoplasty.

REFERENCE

1. Riggins, H. M., and Amberson, J. B., Jr.: Pyogenic Infection of the Pleura Complicating Artificial Pneumothorax, Nat. Tuberc. A. Tr. **31**: 383, 1935.

SELECTIVE THORACOPLASTY FOR PERSISTENT BASAL TUBERCULOUS CAVITIES*

S. O. Freedlander, M.D.
Cleveland, Ohio

SELECTIVE upper thoracoplasty has become the procedure of choice for closing upper lobe tuberculous cavities which have not responded to other forms of treatment. The removal of the entire bony framework covering the lesion, plus one or two ribs below, by increasing the circumferential relaxation and limiting the vertical collapse, has proved most effective in promoting healing of the involved portions of the lung while conserving the uninvolved part. To my knowledge this principle has not been applied to isolated lower lobe tuberculous lesions. Suitable cases are not frequently encountered because lower lobe lesions are usually not isolated, and most of them respond to artificial pneumothorax or phrenic nerve operation. The proper case should have a cavity confined to the lower lobe with the remainder of the lung free from disease. More conservative measures should have had a fair trial. The operation would probably be most effective if the cavity were posterior, since theoretically the large diameters of the lower thorax would seem to make the possibility of an effective collapse remote if the cavity were centrally located. On the left side there might be an objection to uncovering part of the cardiac area. Both of the cases herein reported were on the right. The procedure was to remove the ribs below a horizontal plane two ribs above the lesion. They were removed from the spine almost to the sternum in multiple stages beginning below and posteriorly. The deformity was definite but not unsightly when the patient was clothed.

CASE REPORTS

Case 1.—E. L., white female, age 23 years, was admitted August 21, 1936, to the Lowman Pavilion of Cleveland City Hospital. The patient had developed a persistent cough in September, 1932, and a diagnosis of tuberculosis of the right lung was made. Right pneumothorax was started in May, 1933, by her private physician. In September, 1935, the patient exhibited symptoms of cerebral air embolism following a refill, after which the pneumothorax treatment was discontinued by the mutual desire of patient and physician. Tubercle bacilli persisted in the sputum, and roentgenograms showed a large cavity in the posterior portion of the right lower lobe. The remainder of the lung was clear. The right phrenic nerve was crushed on December 16, 1935. In spite of a good rise in the diaphragm, the large cavity and positive sputum persisted. It was decided to do a limited lower stage tho-

*Read (by title) before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1, 2, 1937.

From the Department of Surgery of Western Reserve University, and Thoracic Service of Cleveland City Hospital.

racoplasty on the right. On October 31, 1936, the first stage was done through a posterior incision. Fifteen cm. of the tenth rib, 17.5 cm. of the ninth rib, and 19.5 cm. of the eighth rib were removed together with the transverse processes. On November 18, 1936, the second stage was performed through an oblique incision, and a total of 67 cm. of the fourth, fifth, sixth and seventh ribs was resected. On December 26, 1936, through a posterior incision 8 cm. of the sixth rib and 15 cm. of the seventh rib were removed.

Fig. 1. Fig. 2.

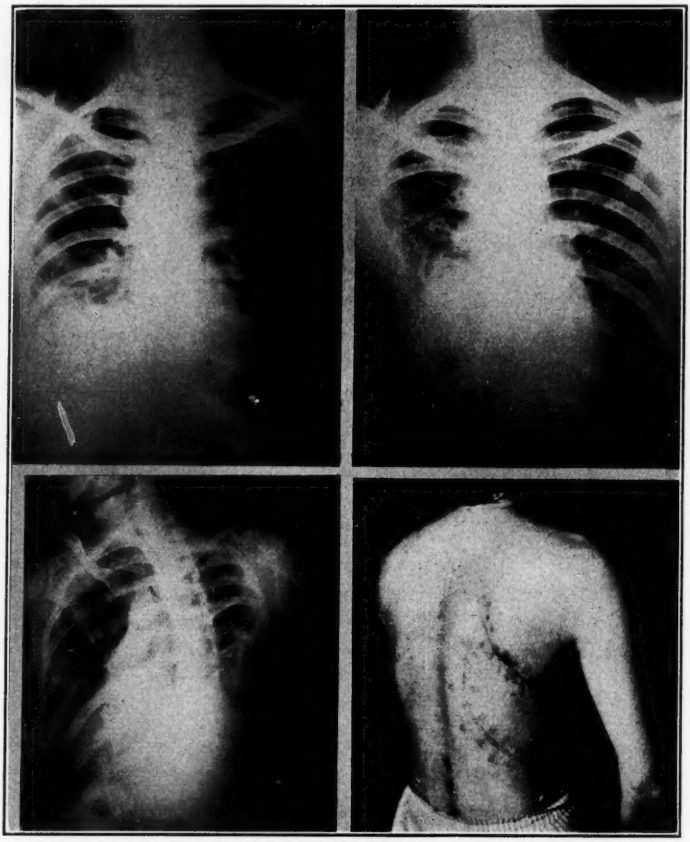


Fig. 3. Fig. 4.

Fig. 1.—Case 1, one year after right phrenic nerve operation. Cavity at right base.
Fig. 2.—Case 1, four months after lower thoracoplasty.
Fig. 3.—Case 1, oblique view after lower thoracoplasty.
Fig. 4.—Case 1, after lower thoracoplasty.

Since the last operation the patient has remained sputum free and gained considerable weight, and roentgenograms in various planes show no evidence of the cavity.

CASE 2.—E. E., colored male age 29 years, was admitted to the Lowman Pavilion of Cleveland City Hospital February 10, 1933, with a history of persistent cough

for three months, a moderate amount of mucopurulent expectoration, seventeen pound weight loss, night sweats, and some pain in the right chest.

Physical examination showed evidence of partial consolidation of the right lower lobe. The sputum contained tubercle bacilli. A roentgenogram showed dense infiltration of the right lower lobe. Pneumothorax treatment was attempted but no pleural space could be found. Serial films showed resolution with excavation, and a large cavity became apparent in the right lower lobe; the remainder of the lung field remained fairly clear. On April 7, 1934, a permanent interruption of the right phrenic nerve was done. The diaphragm became elevated and stationary, and the patient's general condition became much improved but the cavity persisted. The sputum was much diminished in amount but still showed tubercle bacilli. Lateral roentgenograms showed the cavity to be posterior, and it was decided to do a selective

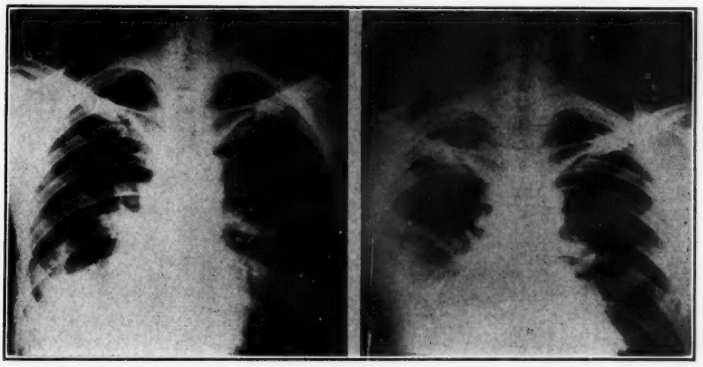


Fig. 5. Fig. 6.

Fig. 5.—Case 2, after phrenic nerve operation. Large cavity right base.
Fig. 6.—Case 2, after lower thoracoplasty.

lower thoracoplasty in multiple stages. On March 20, 1935, portions of the ninth, tenth and eleventh ribs were removed by subperiosteal resection; 26 cm. of the ninth, 24 cm. of the tenth, and 16 cm. of the eleventh. On April 10, 1935, 24 cm. of the eighth rib, 25 cm. of the seventh rib, and 27 cm. of the sixth rib were resected including the transverse processes. On May 8, 1935, through an axillary incision, 7.5 cm. of the anterior portion of the seventh rib and cartilage were removed, 6 cm. of the sixth rib and cartilage, 11 cm. of the fifth rib and cartilage, 10 cm. of the fourth rib and cartilage, and 6 cm. of the third rib and cartilage.

The postoperative course was uneventful. The patient became sputum free and gained weight. Recent films have shown no residual cavitation, while the upper part of the right lung and the entire left lung have remained clear.

In Memoriam

David Alexander Stewart

The members of the American Association for Thoracic Surgery mourn the loss of an active member and friend in the person of David Alexander Stewart of Ninette, Manitoba.

Dr. Stewart entered antituberculosis work immediately after graduation in medicine, and as medical superintendent of the Manitoba Sanatorium was the director of antituberculosis work in his own province, and one of the leaders in antituberculosis thought in Canada for the past twenty years. Dr. Stewart filled every office of honor and distinction that could be conferred upon him by the tuberculosis workers in Canada. During his lifetime he developed a very excellent institution in Manitoba for tuberculosis therapy, and carried on a most active preventive program.

Dr. Stewart was endowed with almost unlimited energy, a most active mind, and vivid imagination. He saw realities as they were and as they had been, and visualized the course of things for the future. This gave him a prophetic as well as a realistic outlook, and his literary skill enabled him to express strikingly what he saw.

These characteristics early established him as the interpreter of tuberculosis knowledge of the day and as the great educator who was able to impress upon students his ideas for the prevention of tuberculosis, and to promote an understanding of the disease among the masses of the people.

The loss of his stimulating personality and his aggressive leadership will be felt by every group of tuberculosis workers across Canada, but perhaps the greatest loss of all is his literary skill in interpreting clearly and forcibly the antituberculosis movement to the masses of the people.

R. G. Ferguson.

Book Review

THE COLLAPSE THERAPY OF PULMONARY TUBERCULOSIS. By John Alexander, Professor of Surgery, University of Michigan, and Surgeon-in-Charge, Division of Thoracic Surgery, Department of Surgery, University of Michigan Hospital. Charles C. Thomas, Springfield, Illinois, and Baltimore, Maryland, 705 pages.

When John Alexander's book, "The Surgery of Pulmonary Tuberculosis," appeared thirteen years ago, the American medical profession had available for the first time a review of what had already been accomplished by the application of surgical methods to the treatment of pulmonary tuberculosis. There can be no doubt that it exerted a potent influence in awakening the phthisiologists of the country to the enormous help which surgical measures can render in properly selected cases. Since that time our knowledge of the whole question has grown so much that it is no longer necessary to defend the principle of the use of surgical methods. Whatever controversies there are now are concerned rather with the question of what form of surgical therapy should be used in a particular case and with the increasing extension of the indications for surgical attack. We, who are interested in thoracic diseases, have long awaited the second edition of Alexander's book. We have realized that no one could write more authoritatively on the subject than the distinguished author of that book. This reviewer was among many who expected a masterpiece. The result fulfills our expectations.

For the most part the book has been written by Alexander himself, but important contributions have been made by others; for example, two chapters on the Physiological Principles and Pathology of Pulmonary Collapse by Max Pinner, two chapters on Pneumothorax by John Blair Barnwell, and one chapter on Oleothorax by Kirby Smith Howlett, Jr. Also, in the preface the author graciously acknowledges his indebtedness to his former pupils whose names he lists, many of whom have now become distinguished thoracic surgeons. The book begins with a perspective of the collapse therapy of pulmonary tuberculosis in which the results of that principle of treatment combined with sanatorium treatment are clearly shown, by a statistical study, to be far better than those of sanatorium treatment alone. Fortunately the need of this sort of education of the phthisiologists is not nearly so great now as it was ten years ago. The next chapter on the evolution of surgical therapy is an excellent historical review of the subject. Other chapters cover all the many phases of collapse therapy. With amazing skill in succeeding chapters Alexander has handled the innumerable details of the different methods of collapse therapy. Operative technique both by description and by illustration is so well given that the reader can have no doubt about the smallest point. The exhaustiveness with which the literature

has been reviewed is demonstrated by the bibliography of 1,342 references.

The large personal experience of the author commands respect for his views, and for that reason he may be pardoned for occasional statements which may seem to others as perhaps too dogmatic and at times almost *ex cathedra*. For example, on page 473 he designates the incisions which he prefers for thoracoplasty as "proper incisions" and lists others as "faulty." The reviewer, for the last few years, has employed an incision the upper limit of which is considerably lower than the one designated by Alexander as "proper." It gives better cosmetic results without sacrifice of excellent exposure of the first rib. A good cosmetic result ought to be a matter of considerable importance, especially in women. The reviewer notes also with some astonishment that Alexander prefers nitrous oxide and oxygen anesthesia. If general anesthesia is to be used, the advantages of cyclopropane, both theoretical and practical, over other forms of inhalation anesthetics seem to the reviewer to be outstanding. There are some other points in which many will disagree with the author, but for the most part they are small points which do not detract from the superlative excellence of the book as a whole.

Some readers will perhaps be amazed that only a footnote of five lines (page 378) is devoted to the Semb operation. Alexander, however, has rightly shown that many others had previously used the principle of apicolysis with thoracoplasty by severing Sibson's fascia and the strands of connective tissue that suspend the apex to the tubercles of the cervical vertebrae, cervical nerve roots and other structures of the neck. If the first rib is removed in its entirety, or even anterior to the scalene tubercle, it is about as easy to remove much of the periosteum as to leave it. Doubtless many surgeons, therefore, for many years have been carrying out the essential principles of the Semb operation. At least the reviewer has.

From a technical standpoint the book maintains the high standard set in other books by Thomas. There are exceptionally few typographical errors. The illustrations are excellently reproduced, the paragraph headings make for easy reference, and the index seems complete. It is interesting, however, that for some unexplained reason the first 106 pages are in double columns. To the reviewer the format of the rest of the book is more pleasing.

This book of necessity will be a bible to all those who are interested in pulmonary tuberculosis. It should be read as widely by those who are not surgeons as by those who carry out the techniques of collapse therapy. It is hoped also that it may be a deterrent to those casual operators who, unversed in a sound knowledge of tuberculosis and ignorant of the indications for and the technique of thoracoplasty, undertake occasionally the removal of ribs in the belief that they are applying modern therapy to a patient unfortunate enough to be under their care.

Evarts A. Graham.

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The American Association for Thoracic Surgery
Atlanta, Georgia, April 4, 5, 6, 1938

Program

MONDAY, APRIL 4

9:00 A.M. Business meeting. Atlanta Biltmore Hotel.

9:30 A.M. Scientific session. Atlanta Biltmore Hotel.

1. Treatment of Acute Empyema Thoracis by Open Intercostal Drainage.
   Joseph Weinberg, Omaha, Neb.
2. Beck's Operation. Report of Two Cases in Which It Was Used.
   A. L. Lockwood, Toronto, Canada.
3. Wounds of the Heart.
   I. A. Bigger, Richmond, Va.
4. Carcinoma of the Thoracic Esophagus: Experimental Investigation and Report of Successful Resection With Anastomosis of the Stomach With the Esophagus.
   W. E. Adams and (by invitation) D. Phemister, Chicago, Ill.
5. Upper Esophagostomy: Its Indications and Uses.
   Carl Eggers, New York.

2:00 P.M.

6. Observations on Mediastinal Tumors: A Study of Fifty-Two Cases Excluding Aneurysms and Substernal Thyroids.
   Evarts A. Graham and (by invitation) Brian Blades, St. Louis, Mo.
7. Aspiration Biopsy of the Lung.
   J. Samuel Binkley (by invitation), New York.

3:15 P.M. Barbecue, Druid Hills Club. Buses leave Hotel Biltmore (members and guests invited).

TUESDAY, APRIL 5

9:00 A.M. Atlanta Biltmore Hotel.

8. Results of Thoracoplasty Using Extensive Costal Resection Over a Period of Seven Years. Study of Operative and End-Results in 500 Cases.
   Edward J. O'Brien and (by invitation) J. C. Day, Detroit, Mich.
9. Tension Cavities of the Tuberculous Lung. Mechanics, Pathology, and Management. A Clinical and Experimental Study.
   Pol N. Coryllos and George Ornstein, New York.
10. Extrapleural Thoracoplasty. Further Experiences With the Multiple Stage Muscle-Splitting Operation.
    Jerome Head, Chicago, Ill.
11. Thoracoplasty for Tuberculosis and Chronic Empyema Through Short Incisions: Experiences With a New Method of Rib Removal.
    Owen H. Wangensteen, Minneapolis, Minn.
12. Results and Complications of the Apicolysis Thoracoplasty.
    Joseph Gale, Madison, Wis.
13. Myoplastic Thoracoplasty.
    Ethan F. Butler, Ithaca, New York.

2:00 P.M. Executive session.

2:30 P.M. Presidential Address. Hiatus Hernia of the Diaphragm.
Stuart W. Harrington, Rochester, Minnesota.

14. Extrapleural Pneumothorax.
    Ronald Belsey, London, England (by invitation). Introduced by J. E. H. Roberts, London, and Edward D. Churchill, Boston, Massachusetts.
15. Lobectomy and Pneumonectomy in Pulmonary Tuberculosis.
    John Jones, Los Angeles, Calif.
16. Tuberculosis of the Large Bronchi. H. McLeod Riggins, New York.

7:30 P.M. Annual dinner. Atlanta Biltmore Hotel.

WEDNESDAY, APRIL 6

9:00 A.M. 17. Bronchopleural Fistulae Complicating Pulmonary Tuberculosis.
Oscar Auerbach and Samuel Lipstein (by invitation), New York.

18. Operative Correction of Pectus Excavatus.
    Alton Ochsner, New Orleans, La.
19. Late Results of Lobectomy for Bronchicetasis in Children.
    John V. Bohrer, New York.
20. Mechanics of Pulmonary Abscess and Bronchiectasis With Suggested Method of Treatment.
    Minas Joannides, Chicago, Ill.
21. Cinéfluoroscopic Studies of Peculiar Breathing and Chest Motion.
    Otto C. Pickhardt, William H. Stewart, Grant Thorburn, New York.

AFTERNOON. Arrangements for golf and for skeet shooting.